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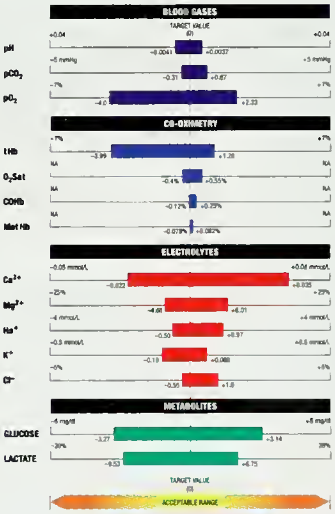

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CONTENTS

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EDITORIALS

Tracheal Intubation: Training and Retention
by Thomas A Barnes—Boston, Massachusetts 227

Radial Arterial Lines and Sticks: What Are the Risks?
by Charles G Durbin Jr—Charlottesville, Virginia 229

Dual Control Modes, Closed Loop Ventilation, Handguns, and Tequila
by Richard D Branson—Cincinnati, Ohio 232

ORIGINAL CONTRIBUTIONS

Recertification of Respiratory Therapists' Intubation Skills One Year After Initial Training: An Analysis of Skill Retention and Retraining
by Michael J Bishop, Piotr Michalowski, John D Hussey, Lewis Massey, and S Lakshminarayan—Seattle, Washington 234

The Impact of Turnover among Respiratory Care Practitioners in a Health Care System: Frequency and Associated Costs
by James K Stoller, Douglas K Orens, and Lucy Kester—Cleveland, Ohio 238

Assessment of Aspiration in Patients with Tracheostomies: Comparison of the Bedside Colored Dye Assessment with Videofluoroscopic Examination
by William T Peruzzi, Jerilyn A Logemann, Donna Currie, and Suellen G Moen—Chicago, Illinois 243

Heliox Delivery with Noninvasive Positive Pressure Ventilation: A Laboratory Study
by Sunisa Chatmongkolchart, Robert M Kacmarek, and Dean R Hess—Boston, Massachusetts 248

CASE REPORTS

Patient-Ventilator Interactions during Volume-Support Ventilation: Asynchrony and Tidal Volume Instability—A Report of Three Cases
by Thierry M Sottiaux—Gosselies, Belgium 255

Weaning to Extubation Directly from High-Frequency Oscillatory Ventilation in an Infant with Cystic Lung Disease and Persistent Air Leak: A Strategy for Lung Protection
by Lori Seller, Krishna Mullahoo, Stephen Liben, and Larry C Lands—Montreal, Quebec, Canada 263

SPECIAL ARTICLES

Innovations in Mechanical Ventilation: What Are (and What Should Be) the Drivers?
by Neil R MacIntyre—Durham, North Carolina 267

LETTERS

Device Evaluation: A Critique
by Robert L Chatburn—Cleveland, Ohio 273

Allen's Test: Fact or Myth?
by Harold A Lanni—Bay Shore, New York, and Stephen G Smith—Stony Brook, New York 274

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CONTENTS

CONTINUED

Aerosols and the Profession of Respiratory Care: Leading the Way Out of the Fog
by Kenneth E Noblett—Evansville, Illinois **275**

The Lack of Data on Off-Label Use of Noninvasive Positive Pressure Ventilation in Children
by Thomas Serrano—Piscataway, New Jersey **276**

BOOKS, FILMS, TAPES, & SOFTWARE

Fraser and Paré's Diagnosis of Diseases of the Chest, 4th ed (4 volumes) (Fraser RS, Colman N, Müller NL, Paré PD)
reviewed by Cornelia Dahm—Seattle, Washington **278**

Comprehensive Respiratory Medicine (Albert RK, Spiro SG, Jett JR, editors)
reviewed by Peter W Bates—Portland, Maine **279**

Textbook of Respiratory Medicine, 3rd ed (2 volumes) (Murray JF & Nadel JA, editors)
reviewed by Derel Finch—Seattle, Washington **280**

Textbook of Critical Care, 4th ed (Grenvik A, senior editor; Ayres SM, Holbrook PR, Shoemaker WC, editors)
reviewed by George N Giacoppe—Seattle, Washington **281**

Clinical Practice in Respiratory Care (Fink JB & Hunt GE, editors)
reviewed by David W Chang—Athens, Georgia **282**

ALSO IN THIS ISSUE

285 AARC Membership Application

224 Abstracts from Other Journals

296 Advertisers Index & Help Lines

296 Author Index

287 Call for OPEN FORUM Abstracts

294 Calendar of Events

289 Manuscript Preparation Guide

293 New Products

295 Notices

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Incidence of ARDS—Roupie E. Intensive Care Med 2000 Jun;26(6):816-817.

Protecting the Public Health by Strengthening the Food and Drug Administration's Authority over Tobacco Products—Myers ML. N Engl J Med 2000 Dec 14;343(24):1806-1809.

Drug Therapy: Prevention and Treatment of Influenza (review)—Couch RB. N Engl J Med 2000 Dec 14;343(24):1778-1787.

Another Perspective on SUPPORT—Luce JM. Am J Med 2000 Dec 1;109(8):686-687.

Helium/Oxygen and Severe COPD—Morice AH. Lancet 2000 Nov 25;356(9244):1785-1786.

Smoking and Mental Illness: A Population-Based Prevalence Study—Lasser K, Boyd JW, Woolhandler S, Himmelstein DU, McCormick D, Bor DH. JAMA 2000 Nov 22/29;284(20):2606-2610.

CONTEXT: Studies of selected groups of persons with mental illness, such as those who are institutionalized or seen in mental health clinics, have reported rates of smoking to be higher than in persons without mental illness. However, recent population-based, nationally representative data are lacking. OBJECTIVE: To assess rates of smoking and tobacco cessation in adults, with and without mental illness. DESIGN, SETTING, AND PARTICIPANTS: Analysis of data on 4411 respondents aged 15 to 54 years from the National Comorbidity Survey, a nationally representative multistage probability survey conducted from 1991 to 1992. MAIN OUTCOME MEASURES: Rates of smoking and tobacco cessation according to the number and type of psychiatric diagnoses, assessed by a modified version of the Composite International Diagnostic Interview. RESULTS: Current smoking rates for respondents with no mental illness, lifetime mental illness, and past-month mental illness were 22.5%, 34.8%, and 41.0%, respectively. Lifetime smoking rates were 39.1%, 55.3%, and 59.0%, respectively ($p < 0.001$ for all comparisons). Smokers with any history of mental illness had a self-reported quit rate of 37.1% ($p = 0.04$), and smokers with past-month mental illness had a self-reported quit rate of 30.5% ($p < 0.001$) compared with smokers without mental illness (42.5%). Odds ratios for current and lifetime smoking in respondents with mental illness in the past month vs respondents without mental illness, adjusted for age, sex, and region of the country, were 2.7 (95% confidence interval [CI], 2.3-3.1) and 2.7 (95% CI, 2.4-3.2), respectively. Persons with a mental disorder in the past month consumed approximately 44.3% of cigarettes smoked by this nationally representative sample. CONCLUSIONS: Persons with mental illness are about twice as likely to smoke as other persons but have substantial quit rates.

Bleeding and Pneumonia in Intensive Care Patients Given Ranitidine and Sucralfate for Prevention of Stress Ulcer: Meta-Analysis of Randomised Controlled Trials—Messori A, Trippoli S, Vaiani M, Gorini M, Corrado A. BMJ 2000 Nov 4;321(7269):1103-1106.

Objectives: To determine the effectiveness of ranitidine and sucralfate in the prevention of stress ulcer in critical patients and to assess if these treatments affect the risk of nosocomial pneumonia. Design: Published studies retrieved through MEDLINE and other databases. Five meta-analyses evaluated effectiveness in terms of bleeding rates (A: ranitidine v placebo; B: sucralfate v placebo) and infectious complications in terms of incidence of nosocomial pneumonia (C: ranitidine v placebo; D: sucralfate v placebo; E: ranitidine v sucralfate). Trial quality was determined with an empirical ad hoc procedure. Main outcome measures: Rates of clinically important gastrointestinal bleeding and nosocomial pneumonia (compared between the two study arms and expressed with odds ratios specific for individual studies and meta-analytic summary odds ratios). Results: Meta-analysis A (five studies) comprised 398 patients; meta-analysis C (three studies) comprised 311 patients; meta-analysis D (two studies) comprised 226 patients; and meta-analysis E (eight studies) comprised 1825 patients. Meta-analysis B was not carried out as the literature search selected only one clinical trial. In meta-analysis A ranitidine was found to have the same effectiveness as placebo (odds ratio of bleeding 0.72, 95% confidence interval 0.30 to 1.70, $p=0.46$). In placebo controlled studies (meta-analyses C and D) ranitidine and sucralfate had no influence on the incidence of nosocomial pneumonia. In comparison with sucralfate, ranitidine significantly increased the incidence of nosocomial pneumonia (meta-analysis E: 1.35, 1.07 to 1.70, $p=0.012$). The mean quality score in the four analyses (on a 0 to 10 scale) ranged from 5.6 in meta-analysis E to 6.6 in meta-analysis A. Conclusions: Ranitidine is ineffective in the prevention of gastrointestinal bleeding in patients in intensive care and might increase the risk of pneumonia. Studies on sucralfate do not provide conclusive results. These findings are based on small numbers of patients, and firm conclusions cannot presently be proposed.

Association between Acute Hypobaric Hypoxia and Activation of Coagulation in Human Beings—Bendz B, Rostrup M, Sevre K, Andersen TO, Sandset PM. Lancet 2000 Nov 11;356(9242):1657-1658.

The risk of venous thrombosis is thought to be increased by flying. In a study of 20 healthy male volunteers who were suddenly exposed to a hypobaric environment similar to that encountered within aeroplane cabins, markers of activated coagulation transiently increased by two-fold to eight-fold. We suggest that hypobaric hypoxia, with sedentariness and dehydration, may cause this increased risk of venous thrombosis.

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Airway Management and Direct Laryngoscopy: A Review and Update—Levitan R, Ochroch EA. Crit Care Clin 2000 Jul;16(3):373-388, v.

Direct laryngoscopy is the direct visualization of the larynx while using a rigid laryngoscope to distract the structures of the upper airway. This article reviews the anatomy relevant to laryngoscopy and then presents a stepwise approach to the procedure. Alternative intubation techniques, positioning, laryngoscopy blades, and stylets are then covered. Pharmacologic adjuncts are discussed briefly as they relate to the difficult airway and incorporation into overall airway management.

Emergent Management of the Airway: New Pharmacology and the Control of Comorbidities in Cardiac Disease, Ischemia, and Valvular Heart Disease—Horak J, Weiss S. Crit Care Clin 2000 Jul;16(3):411-427.

Once it is decided that the patient in distress requires tracheal intubation, the primary goal is to secure the airway as quickly and safely as possible to assure adequate oxygenation and ventilation. The clinician should quickly review the patient's history, physical examination findings, and laboratory data to determine the presence of cardiovascular disease, assess intravascular volume status, and formulate a plan for induction of anesthesia. The stresses of hypoxia, hypercarbia, acidosis, and extreme fatigue result in near-maximal sympathetic outflow that is manifest as tachycardia, labile blood pressure, and increased myocardial contractility. The astute clinician should anticipate that the tachycardia and hypertension associated with laryngoscopy and tracheal intubation is followed by a period of hypotension. This postintubation hypotension results from the acute marked attenuation of the sympathetic tone associated with resolution of hypoxia and hypercarbia, direct drug-induced negative inotropic effect, and vasodilation. The decrease in sympathetic vascular tone may result in hypotension by exacerbating the decrease in cardiac preload and afterload from hypovolemia. In addition, the use of positive pressure ventilation and positive end-expiratory pressure (PEEP) in these hypovolemic patients will further decrease ventricular preload by impeding venous return, leading to profound hypotension. Several pharmacologic agents are required to treat effectively the hemodynamic perturbations associated with induction, laryngoscopy, and tracheal intubation. Most sedative hypnotic agents that are administered for induction provide minimal to no analgesia. Patients are most often given a combination of drugs to provide adequate sedation, analgesia to blunt the noxious stimuli, and muscle relaxation to facilitate the laryngoscopy. The major challenge is to choose a combination of drugs that at the appropriate doses, effectively blunt the responses to intubation without contributing to postlaryngoscopy hypotension. One can use several strategies to accomplish these goals; administration of a narcotic analgesic before induction decreases the dose of induction agent and can attenuate the sympathetic response to intubation. Because of the prevalence of cardiovascular disease and hypovolemia in this population of patients, all chosen drugs should have minimal negative effect on cardiac function and patients with hypovolemia should be hydrated. Most clinical studies have been performed in hemodynamically stable patients, so the routine dosages of sedative hypnotics should be reduced substantially and titrated to effect. An additional strategy is to treat significant hemodynamic perturbations with vasopressors, vasodilators, short-acting selective β_1 blockers, and inotropic agents. The choice of vasoactive agent depends on the magnitude of the hemodynamic response and the presence of specific underlying cardiovascular pathology.

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Tracheal Intubation: Training and Retention

In this issue of RESPIRATORY CARE, Bishop et al[1] report on the retention of tracheal intubation skills by 11 respiratory therapists (RTs) one year after they received training in airway management. The paper is an important contribution to an area where little scientific evidence exists. Infrequent performance of intubation by basic emergency medical technicians has been shown to decrease the intubation success rate.[2,3] Bishop et al report that an RT with a record of several intubations had poor skill retention one year after initial training. The report raises several important questions:

SEE THE ORIGINAL STUDY ON PAGE 234

How many intubations per year are needed to retain airway management skills?

What type of airway management training and certification program should be in place for RTs?

What conclusions can be drawn from the data on skill retention and retraining intervals?

What level of supervision and record keeping should be in place?

What is the role of alternative airways in hospitals where health care providers experienced in tracheal intubation are not available?

These questions and the discussion of the importance of cognitive knowledge and the identification of critical errors will be of interest to health care professionals involved with airway management training and supervision.

Large medical centers have more RTs available to provide airway management and present a different scenario than community hospitals. In 1991, the Duke University Medical Center (Duke) intubation service staffed by registered RTs (RRTs) had a 95% (791/833) success rate (≤ 3 attempts) and a complication rate of 12% (96/833).[4] A review of 1999 data from Duke shows a 95% (901/951) success rate (≤ 3 attempts) and a complication rate of 3% (29/951) (Thalman JL, Duke University Medical Center, Durham, North Carolina, Dec 18, 2000, personal communication). Complication rate was based on the incidence of oral bleeding, vomiting, aspiration, bradycardia, or broken tooth. The intubation service at Duke was established in 1976 and operates under a detailed Respiratory Care Service airway management policy for certification and retraining. The policy requires RRT, advanced cardiac life support (ACLS), and pediatric advanced life support (PALS) credentials as prerequisites for receiving advanced airway certification. "Airway certified" means that the RRT is able, independently if necessary, to perform intubations and tube changes. The training policy requires a minimum score of 85% on a 130-question written advanced airway management examination. Practice and skill checks on manikins replace operating room (OR) experience. However, the final step to receive Duke advanced airway certification is the completion of 10 successful intubations of patients outside the OR under the direct observation of a supervisor or clinical instructor. To maintain airway certification at Duke, and avoid retraining, RTs must perform a minimum of 10 successful intubations per year without complications. Two RRTs and a physician are usually present for tracheal intubations outside the OR. If the first RRT intubation attempt fails, the second attempt is completed by the other RRT on the team.

Medical centers with a smaller number of acute care medical and surgical beds provide fewer opportunities for RRTs to intubate. Respiratory therapy departments in some hospitals may have only a few RRTs who hold both ACLS and PALS certification. These circumstances of limited experience and no advanced training may lead to a poor intubation success rate and a high incidence of complications. Studies of basic emergency medical technicians completing 9 to 10 hours of training and a limited number of intubation encounters report poor success rates, ranging from 49% to 53%.[2,3] Paramedics, flight nurses, and RTs with advanced training, multiple encounters, and tight supervision have a success rate of > 90%.[4–8] A 12-month study of a community hospital emergency tracheal intubation program reported an RT success rate of 96% (71/74) and a complication rate of 18% (13/74), versus a success rate of 57% (39/69) and complication rate of 57% (39/69) among medical doctors and certified registered nurse anesthetists.[7]

The American Heart Association's new Guidelines 2000 for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care state:

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> When tracheal intubation is attempted by providers with insufficient skill, the following complications may be seen: trauma to the oropharynx, ventilation withheld for unacceptably long periods, delayed or withheld chest compressions, esophageal or bronchial intubation, failure to secure the tube, and failure to recognize misplacement of the tube. Therefore, inexperienced providers should use only those airway management devices for which they are adequately trained. Those who perform tracheal intubation require either frequent experience or frequent retraining. EMS [emergency medical service] systems should keep a record for each provider, documenting the number of intubations performed, as well as the success rates and complications.[8]

Long-term retention of airway management skills may be negatively impacted by ACLS courses that are too long and have inadequate time for skill practice.[9] An evaluation of ACLS-trained medical residents during actual in-hospital resuscitations recorded improper cardiopulmonary resuscitation performance, poor team leadership and patient assessment, difficulties with equipment, and failure to follow the recommended American Heart Association algorithm.[9]

Well-trained paramedics can successfully perform emergency uncomplicated tracheal intubations during in-hospital cardiopulmonary resuscitation.[10] The need for frequent intubation experience for staff in small hospitals might be addressed by employment of part-time paramedics or RRTs who work part-time as paramedics. Another approach might be to hire part-time RRTs who receive intubation experience in a larger hospital and specifically designate them as airway managers. Frequent (monthly) rotations of RRTs to the OR for airway management training may be a solution for some community hospitals.

Alternative airway devices such as the laryngeal mask airway (LMA) or the esophageal tracheal combitube are acceptable airway devices that may be useful alternatives to tracheal intubation.[11,12] During attempts to ventilate the lungs of a patient in a cardiac arrest, the LMA and esophageal tracheal combitube are better alternatives to the face mask. To achieve good outcomes with these devices, health care providers must be well trained in their use and maintain their knowledge and skills through frequent practice and clinical use.[8] In comparison with the tracheal tube, the LMA provides equivalent ventilation.[12] Training in the placement and use of an LMA is simpler than tracheal intubation because laryngoscopy and visualization of the vocal cords are not necessary for insertion of the LMA.[8]

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Radial Arterial Lines and Sticks: What Are the Risks?

Invasion of the arterial circulation is a very common intervention in critically ill patients, second in frequency only to intravenous cannulation.[1] The need for frequent arterial blood gas measurements and continuous blood pressure monitoring are the usual cited reasons for arterial cannulation.[2,3] With every medical procedure there are associated risks. Estimating these risks and weighing the presumed benefits from the procedure, as well as considering the risks of not performing the intervention, allow caregivers to make reasonable decisions about when to place an arterial line. Do we have accurate data about the risks and benefits of arterial sticks and cannulations? Though we have some information about risks and complications of arterial cannulation, there are few data about the benefits of this form of monitoring. This lack of information about the benefits of monitoring is not confined to arterial lines; there is almost no information supporting the value of any form of monitoring. However, expert opinion and usual "standards of care" suggest that these monitors have value and should be selectively used based on the expected benefits and known risks in individual patients.

See The Letter on Page 274

The radial artery is the most frequent site of arterial access. This is because of its superficial and predictable location, minimal pain on penetration, ease of cannulation, convenience of securing a cannula, and presence of abundant collateral circulation at the wrist and in the digits. On the other hand, hemodynamic measurements are often affected by wave artifacts, which are greater in the radial artery than in more central arteries. Radial arterial invasion carries several general types of risk:

1. Vascular thrombosis
2. Distal embolization
3. Proximal embolization
4. Vascular spasm
5. Skin necrosis over catheter site
6. Line disconnection and bleeding
7. Accidental drug injection
8. Local infection
9. Systemic infection
10. Damage to nearby structures

The first five of those concerns are related to obstruction of arterial blood flow. Since radial cannulation was described by Barr in 1961,[4] clinicians have worried about arterial thrombosis from indwelling catheters leading to hand ischemia and infarction. Similar concerns have been raised with regard to arterial puncture, and it is generally recognized that the arterial pulse is often lost following puncture. This is assumed to be because of arterial spasm, less often because of thrombosis, and is usually transient. It has been hypothesized that, in order to minimize the risk of hand loss should radial occlusion occur, checking for adequate collateral circulation from the ulnar artery is necessary prior to radial cannulation or puncture. More than 97% of patients have adequate flow through collaterals to prevent hand ischemia should either the radial or ulnar artery be interrupted. Described by Dr Edgar Van Nuys Allen in 1929, the so-called "Allen's test" has been used for many years to clinically assess the adequacy of collateral blood flow at the wrist.[5] Dr Allen described this test to document chronic occlusion of the wrist arteries from thromboangiitis obliterans (Buerger's disease), a distal small vessel occlusive disease most often caused by excessive smoking.

Dr Allen, the son of a pathologist, received his MD from the University of Minnesota, completed a fellowship at the Mayo Clinic, and was a post-doctoral student at Leipzig, Germany, when he published his classic clinical description. According to Dr Allen,

> If obstruction of the ulnar artery is suspected, the radial arteries are located by their pulsations; the examiner places one thumb lightly over each radial, with the four fingers of each hand behind the patient's wrist, thus holding the wrist lightly between the thumb and fingers. The patient closes his hands as tightly as possible for a period of one minute in order to squeeze the blood out of the hand; the examiner compresses each wrist between his thumb and fingers thus occluding the radial arteries; the patient quickly extends his fingers partially while compression of the radial arteries is maintained by the examiner. The return of color to the hand and fingers is noted. In individuals with an intact arterial tree the pallor is quickly replaced by rubor of a higher degree than normal, which gradually fades to the normal color. If the ulnar arteries are occluded, pallor is maintained for a variable period. . .[5]

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Though that is the classical description, many variations have been added to this test as it has been employed in entirely different circumstances. Current differences from the original description include testing only one hand at a time, compressing both ulnar and radial arteries manually and then releasing them one at a time, watching for rubor on the distal-most digit, and assigning a positive or negative result based on the time in seconds until filling is seen. A filling time less than 6 seconds is considered a negative Allen's test (normal flow) by some,[6] whereas others consider less than 15 seconds indicative of adequate flow.[7] Other methods used to determine adequate collateral filling include plethysmographic pulse measurement,[8] Doppler flow detector, and digital blood pressure determination when one artery is occluded.[9]

Concern about collateral flow is supported by the high frequency of radial artery thrombosis following cannulation, ranging from 7% to 30% of arteries. Risk factors increasing the frequency of thrombosis include large catheter size, small artery diameter, traumatic insertion, multiple arterial sticks, duration of cannulation, and the type of material of which the catheter is made. The consequences of thrombosis are less clear. Although there are sporadic reports of hand ischemia, these are usually seen in patients with poor cardiac function,[10] patients in shock or receiving high doses of vasopressors, and patients with underlying vascular diseases.[11] These ischemic complications are usually not caused by radial artery thrombosis. Severe ischemic damage from arterial cannulation has been estimated to occur in less than 0.01% of patients.[12] Arterial thrombosis seems to be a self-limited problem, usually unassociated with ischemia and with recannulation occurring within several months.[13] The main concern with thrombosis is in the patient requiring repeated cannulation, in which cannulation sites are used up. Although it has not been formally studied, cannulation of an ulnar artery in the presence of thrombosis of the radial artery probably increases the risk of hand injury.

Is a positive Allen's test a contraindication to radial artery cannulation? One small series of cardiac surgery patients were cannulated following a positive Allen's test indicating poor ulnar flow.[14] None of the 16 patients cannulated suffered ischemic consequences. This group was monitored for a short time (< 3 d) and also had received heparin for cardiopulmonary bypass. Based on this highly selected, small group of patients (probably males) the recommendation to ignore the results of the Allen's test and proceed with cannulation has been put forward. In fact, a positive Allen's test is very rare, probably occurring in less than 1% of patients who have not had previous arterial cannulation. However, in patients who have undergone previous cannulations, the Allen's test provides excellent documentation of arterial thrombosis and should be used to direct catheter placement. In that circumstance, it is probably premature to totally ignore the results of the Allen's test, especially if another artery is available and the cannulation is being used for something other than to manage bypass during cardiac surgery.

To complete the list of other possible injuries, clot emboli can cause distal ischemia in the fingers, and clot (or air) pressure flushed retrograde can enter the cerebral circulation and cause a stroke. Thrombosis or local ischemia of the skin above the artery can create a skin defect that may require debridement and skin grafting. The frequency of these occurrences is not known, but they are probably rare. Catheter colonization leading to systemic infection is often mentioned as a source of important problems, but the actual risk appears to be much lower than that from venous catheters.[15] Proper catheter care should make this a diminishing clinical concern. Local skin infection does complicate insertion occasionally. Failure of the catheter, evidence of thrombosis, or suspicion of infection are indications for removal of the device.

Most serious patient injuries due to arterial cannulation occur because of two problems: unrecognized line separation leading to bleeding, and accidental medication injection leading to hand necrosis. The exact incidence of these terrible complications is unknown because they are generally not reported in the literature.[16] Both are evidence of substandard care or accidents. With the change in the quality and quantity of the medical work force, techniques for preventing these tragedies need to be developed.

The last category of complications includes damage to adjacent structures. Direct neural insertion or bleeding into the palmar sheath can cause pressure injury to the radial or median nerve, with development of carpal tunnel syndrome or other sympathetic mediated pain syndrome.[17–19] Early intervention with sympathetic ganglion (stellate) block may reduce the severity of injury.[20] Hematomas can develop into false aneurysms that may need surgical intervention.[21]

With all of these potential problems related to arterial punctures and cannulation, assessment of benefits must be carefully carried out for each patient. Respiratory therapists often perform arterial punctures and occasionally insert arterial lines.[1] Fortunately, with excellent clinical management, serious problems due to these procedures are very rare.

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Dual Control Modes, Closed Loop Ventilation, Handguns, and Tequila

These pages have often praised technologic advances in mechanical ventilation and at times lamented the lack of scientific evidence accompanying these advances.[1,2] Certainly, we have seen improvements in triggering, monitoring, and safety with the introduction of microprocessor control. Yet new modes seem to proliferate without evidence of need or evidence of efficacy.[3] This age-old problem is both nurtured and decried by the scientific community. Though new modes rarely have a background in physiology (proportional assist and adaptive support ventilation being the exceptions), these new methods are described and tested in the literature as part of normal scientific inquiry. These evaluations, often descriptive studies, fail to answer the truly important question: Is it worth it? I include myself in this group and will not belabor the issues surrounding the cost, duration, and complexity of outcome studies that might seek to find a "superior" mode of ventilation.

Current regulations regarding devices do not require randomized, controlled clinical trials to demonstrate efficacy. This is in stark contrast to the level of testing and evaluation required of a new pharmaceutical. In fact, a new ventilator mode can be introduced without any patient testing at all! All that is required is proof that the mode performs the intended function and that the software has been validated. This can all occur in a bench model, far from the nearest patient.

That being the case, clinicians are often left to determine the usefulness of these new techniques. Perhaps most distressing is the fact that the individuals performing most of the education surrounding these new modes are manufacturers' representatives. At best it should be said that they have a perceived conflict of interest.

In this issue of RESPIRATORY CARE, Sottiaux[4] reports a case series of patients developing serious dyssynchrony during volume-support ventilation (VSV). VSV is not really new by today's standards, having been introduced in the mid 1990s. Yet the report by Sottiaux represents only the second published report evaluating this mode.[5] In fact, in my opinion, VSV is not new at all, but represents a modified version of mandatory minute volume, which was introduced by Hewlett in 1976.[6] The initial description by Hewlett used additional mandatory breaths to maintain a constant minute volume. However, the Hamilton Veolar, introduced in 1984, used a version of mandatory minute volume wherein pressure support level increased or decreased based on an 8-breath rolling average to ensure constant minute volume.[7] The operation of VSV simply modifies this technique to change the pressure support level on a breath-to-breath basis to maintain tidal volume (V_T) constant, although, as Sottiaux shows, VSV sometimes reverts to being a minute volume controller.[4]

SEE THE CASE REPORT ON PAGE 255

The Sottiaux case series highlights several areas of concern related to both pressure-support ventilation (PSV) and closed loop control of mechanical ventilation. The first relates to the issue of neural-mechanical dyssynchrony during PSV. Several authors have shown that patients with chronic obstructive lung disease on PSV frequently activate expiratory muscles to end inspiration.[8–11] This phenomenon results from the ventilator inspiratory time being longer than the patient's neural inspiratory time. As Sottiaux has shown nicely here, this problem is exacerbated by a ventilator such as the Siemens Servo 300, which uses a late flow cycle criterion set at 5% of the initial peak flow. One way of alleviating that problem is to decrease the pressure support. However, in the case described, VSV further confounds the situation. This occurs when the VSV algorithm increases the pressure support level, increasing V_T, prolonging inspiration, and further distorting the exhaled-volume/expiratory-time relationship. The end result is air trapping (intrinsic positive end-expiratory pressure), difficulty triggering, and dyssynchrony. This is of course the direct opposite of the intention of the mode.

Another method of alleviating neural-mechanical dyssynchrony is through adjustment of the cycle criterion (unfortunately often called expiratory trigger sensitivity). In many new ventilators the cycle criterion can be adjusted from 10% to 40% of the initial peak flow. In chronic obstructive pulmonary disease, a short inspiratory duration, flow cycle at 40% of the initial flow, might be helpful in reducing dyssynchrony. However, even this is a manual adjustment, and the changing demands of the patient might require a more flexible and responsive system. Du and Yamada have introduced an automated method for choosing cycle criteria.[12] This closed

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loop technique might be combined with PSV or even VSV to improve the safety and efficacy of either mode.

Despite these problems, I believe closed loop ventilator techniques are part of our future. Even in the absence of randomized, controlled, double-blind, clinical trials, the development of new modes will continue. This will occur because it can. However, as we treat sicker and sicker patients with fewer and fewer personnel and resources, these modes may actually prove to be of some value. The current closed loop techniques like VSV and other dual control modes suffer from over-simplification. Each technique uses one input to control one output. Surely the complexity of the respiratory system, which uses a myriad of inputs, requires more sophisticated systems. Recently, Dojat et al[13] evaluated a technique similar to VSV, but with additional signal inputs. Using a modified Hamilton Veolar, their system used the inputs of V_T, respiratory rate, and end-tidal carbon dioxide to manipulate pressure during PSV. In this technique, changes in V_T, respiratory rate, and end-tidal carbon dioxide can all result in an increase or decrease in PSV. By measuring the pressure generated in the first 100 milliseconds of inspiration ($P_{0.1}$) during traditional and modified PSV they were able to demonstrate reduced periods of excessive work load during the modified PSV technique. In this study, excessive work load was described as duration with a $P_{0.1}$ of > 4 cm H_2O.

Other promising results have been seen with closed loop control based on proportional assist ventilation and adaptive support ventilation.[14–19] In each instance, the use of a sound physiologic background (the equation of motion for the respiratory system for proportional assist ventilation and Otis's optimal work of breathing[20] for adaptive support ventilation), multiple inputs, and multiple outputs both show the promise of future closed loop techniques.

Sottiaux's report highlights the current weakness in the development, implementation, and clinician understanding of closed loop ventilation. We should be vigilant in our evaluation of new techniques and realize that what is good for the postoperative patient is not necessarily good for the chronic obstructive pulmonary disease patient. We should partner with our industry colleagues in development of new techniques, not follow blindly behind them. Finally, we should do as Sottiaux has done and share our findings in the literature.

Rob Chatburn brought to my attention a very applicable cautionary quote to the issue of closed loop ventilation: "A computer lets you make more mistakes faster than any invention in human history. . . with the possible exception of handguns and tequila."

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Recertification of Respiratory Therapists' Intubation Skills One Year After Initial Training: An Analysis of Skill Retention and Retraining

Michael J Bishop MD, Piotr Michalowski MD PhD, John D Hussey MBA RRT,
Lewis Massey, and S Lakshminarayan MD

Allied health personnel and nonanesthesiologist physicians often undergo training in tracheal intubation but then may actually use the skill relatively infrequently. This study assessed retention of skills one year after initial training and identified specific areas of knowledge critical to successful performance of intubation. Eleven respiratory therapists on the staff of a 253-bed hospital, each of whom had been trained one year previously in airway management, were evaluated. Prior to returning to the operating room for skills assessment and recertification, each respiratory therapist took a 21-question written exam. Therapists then went to the operating room and a trained observer (anesthesiologist) monitored the intubations performed to see whether critical steps were followed, while a second observer monitored a checklist of skills performed. The attending anesthesiologist recertified the therapist only when all steps were correctly performed and the intubation was successful. There was a poor correlation ($r = -0.25$, $p > 0.1$) between the number of intubations performed by the therapists for emergencies in the previous year and the number of intubations needed to be recertified. There was a negative correlation ($r = -0.8$, $p < 0.05$) between the score on the written test and the number of intubations required for recertification—a higher score meant fewer intubations were needed to achieve recertification. First-pass success occurred significantly more frequently if all skills tested were performed correctly (50/75 first-pass successes had all skills performed correctly vs 10/28 for failed first-pass, $p < 0.01$). The most common errors were levering the blade on the upper teeth (12/91) and tube not inserted from the right side of the mouth (28/104). When the blade was levered, 8 of 10 intubations failed. When the tube was not inserted from the right side of the face, 6 of 12 failed. The useful findings of this study are: (1) occasional performance of intubation did not ensure skill maintenance; (2) cognitive and procedural abilities correlated, suggesting benefits to study as well as to practical training; and (3) two specific mistakes were associated with a high incidence of failure. *Key words: tracheal intubation, training, skills retention, respiratory therapist, retraining.* [Respir Care 2001;46(3):234–237]

Background

Anesthesiologists often provide training in airway management to other physicians, paramedics, and respiratory therapists (RTs).[1,2] Though anesthesiologists and nurse anesthetists use these skills daily and thus maintain them, daily experience is often not available to reinforce training for allied health professionals. The American Heart Association Guidelines 2000 for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care recommend that "Those who perform tracheal intubation require either frequent experience or frequent retraining."[3] However, that is a consensus recommendation and no supporting data or guidelines for retraining are provided.

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Previous publications have documented the safety and efficacy of tracheal intubation by personnel who perform this task relatively infrequently. However, high failure rates have occurred in some settings because of inadequate training or inadequate ongoing experience.[4–6] In terms of the experience needed initially, the skills needed to perform the majority of intubations are acquired relatively quickly, with the learning curve flattening at approximately 30 intubations.[7]

SEE THE RELATED EDITORIAL ON PAGE 227

Our hospital instituted a program that trained RTs to intubate in cases of cardiac arrest.[2] Initial training was monitored and closely supervised by anesthesiologists and certified registered nurse anesthetists. However, we were aware from studies of advanced cardiac life support algorithms that knowledge retention of rarely-used skills may not be satisfactory.[8,9] One year after initial training in intubation, we began recertification training and prospectively studied skill and knowledge retention of the RTs. The 11 RTs who were recertified had performed emergency and urgent ward intubations in adults. The number performed by any given therapist ranged from 0 to 14 (median = 5).

We hypothesized that (1) performance of intubations by therapists during cardiac arrests would be sufficient for maintenance of skills, (2) tracheal intubation is primarily a motor skill and written examination would not be a useful indicator of skills, and (3) performance of a specified set of discrete steps would result in a higher success rate.

Methods

All RTs took a 21-question written exam prior to returning to the operating room for skills assessment. The test included questions concerning pre-intubation assessment of the airway, airway anatomy, appropriate use of blades, appropriate head position, and other factors related to airway management.

Each therapist then returned to the operating room for anesthesiologist-supervised intubations. One of two trained observers (authors JDH and LM) assessed whether the following steps were carried out: airway assessed for signs of possible difficult intubation, laryngoscope and suction checked, mask ventilation established, scope placed in left hand, right hand used to open mouth, blade placed to displace the tongue to the left, blade pulled rather than levered on teeth, tube placed with tip coming in from the right side, and the hand not in the line of sight. The frequency of first-pass success was then noted. Therapists were recertified by supervising anesthesiologists only when all skills on the checklist appeared to be routinely performed, although a minimum of 5 successful first-pass intubations in 5 different patients was required of all therapists. Supervising anesthesiologists were not told the results of the written tests nor the number of intubations performed by the therapist during the preceding year. Comparisons of proportions were performed using chi-square analysis. Correlations were determined using Pearson's correlation coefficient as determined with the SPSS 9.0 statistical software package (SPSS, Chicago, Illinois).

Results

There was a poor correlation ($r = -0.25$, $p > 0.1$) between the number of intubations performed on the floors during the previous year and the number of intubations needed to be recertified (Fig. 1). However, the individual who had performed the most intubations on the floor required only the minimum of 5, and one of two individuals who had done no intubations since original certification required the most intubations for recertification.

There was a negative correlation ($r = -0.8$, $p < 0.05$) between the score on the written test and the number of intubations required for recertification—a higher score meant fewer intubations were needed to achieve recertification.

First-pass success occurred significantly more frequently if all discrete steps were performed correctly (50/75 first-pass successes had all skills performed correctly vs 10/28 for failed first-pass, $p < 0.01$). The most common errors were levering the blade on the upper teeth (12/91) and failing to insert the tube from the right side of the mouth (28/104). When the blade was levered, 8 of 10 intubations failed. When the tube was not inserted from the right side of the face, 6 of 12 failed.

Discussion

The useful findings of this study are:

1. Occasional performance of endotracheal intubation did not ensure skill maintenance.
2. Factual knowledge of the steps in intubation and procedural abilities correlated, suggesting benefits to study as well as to practical training.
3. Two specific mistakes were associated with a high incidence of failure.

Although there is substantial literature on the success rate of allied health personnel in airway management,[1–2,4–6] few data are available on skill retention and retraining. A study of retraining methods for paramedics in pediatric airway management compared a control group (no retraining) with videotape retraining, self-instruction, and lecture-demonstration retraining, and found substantially better skills with lecture-demonstration than with any of the other 3 methods.[10] However, a literature search revealed

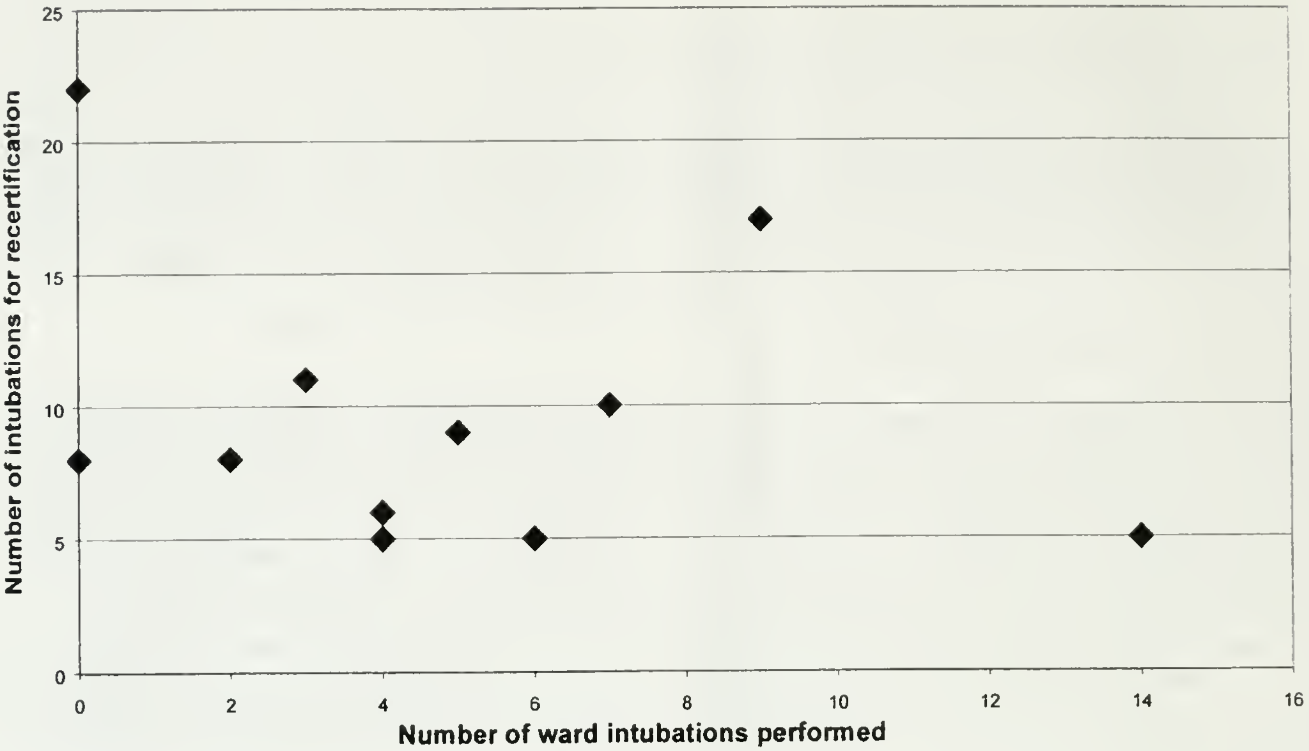

Fig. 1. The number of intubations required for recertification was not well correlated with the number of intubations performed on the ward during the year (r = −0.25, *n* = 11).

extensive information regarding initial training but no other information on retraining in airway management.

In establishing our training program, the need for recertification was based on studies of cardiopulmonary resuscitation training showing that a substantial amount of the knowledge base is gone by 6 months after initial training, and most of it is gone by 2 years. The above-referenced study of pediatric airway management found substantial skills deficits 6 months after initial training, but no information was provided as to the frequency with which intubations had been performed in the field.

A study of the initial learning of endotracheal intubation by anesthesia residents found that 80% success is achieved after 30 intubations and the learning curve then flattens.[7] This figure correlated well with our initial training efforts for RTs, but did not provide information on skill retention or numbers needed for retraining.

We found that performing intubations did not mean that skills would necessarily be retained—at least not uniformly and not with the numbers of intubations performed in this study. We had originally hoped that the performance of 5 intubations over the course of the year would lead to retention of skills. However, only 2 of the 5 therapists who performed 5 or more intubations during the year were sufficiently skilled to recertify with the minimum 5 intubations. One was found to have substantial enough errors in technique that 17 intubations were required prior to recertification.

A power analysis of the data indicates that this degree of correlation might be statistically significant if 60 therapists had been studied and the correlation remained at −0.25. However, such a correlation would not be clinically useful, given the degree of individual variability.

We did find a correlation of test scores with ability to intubate. This has been variable in other studies of education, but may reflect that the test was directly oriented toward the procedure rather than abstract basic knowledge. Specifically, questions asked included where the blade should be placed, appropriate patient position, and airway evaluation.

The conditions at our facility may not be applicable to other facilities, based on the training mix of therapists, the size of the facility, and patients' severity of illness. Our hospital is a 253-bed facility with 145 acute care medical and surgical beds, including 26 intensive care unit beds, a bone marrow transplant unit, and a spinal cord injury unit. Thus, the opportunities for therapist intubations are limited by the number of patients. Larger facilities may offer more intubation experience to individual therapists. The therapists involved included 6 registered RTs, 4 certified therapists, and 1 uncertified therapist. The conclusions of this study apply to these therapists but may not necessarily be applicable in other settings, with therapists with different experience and training.

The study suggests the importance of teaching an algorithmic approach, with special emphasis on appropriate use of the blade and appropriate insertion of the tube, since those were common errors and were associated with failure. Our future retraining will emphasize those two points.

We cannot conclude that our results would apply to therapists who had the opportunity to perform many more intubations in a year. The one therapist who performed the most intubations required only the minimum number of observed intubations to recertify. Perhaps if all of the therapists had performed 14 ward intubations, all would have done equally well. We do know that the one therapist who had performed 9 intubations still required substantial retraining. Given that knowledge, we have decided that we do not know a minimum number that guarantees continued high proficiency, and we plan to continue yearly recertification.

Conclusions

Our experience with recertification of RTs in tracheal intubation leads us to the following conclusions:

1. Infrequent performance of tracheal intubation does not maintain all of the skills needed to perform the task well.

2. Cognitive knowledge does correlate with performance of the task, and we plan to emphasize that prior to annual practical retraining.

3. Emphasis on a pulling rather than a levering action with the blade and on insertion of the tube from the right side rather than from midline should help improve performance.

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The Impact of Turnover among Respiratory Care Practitioners in a Health Care System: Frequency and Associated Costs

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BACKGROUND: Retention of respiratory therapists (RTs) is a desired institutional goal that reflects department loyalty and RTs' satisfaction. When RTs leave a department, services are disrupted and new therapists must undergo orientation and training, which requires time and expense. Despite the widely shared goal of minimal turnover, neither the annual rate nor the associated expense of turnover for RTs has been described. STUDY PURPOSE: Determine the rate of RT turnover and the costs related to training new staff members. METHODS: The Cleveland Clinic Health System is composed of 9 participating hospitals, which range from small, community-based institutions to large, tertiary care institutions. To elicit information about annual turnover among RTs throughout the system, we conducted a survey of key personnel in each of the hospitals' respiratory therapy departments. To calculate the costs of training, we reviewed the training schedule for an RT joining the Respiratory Therapy Section at the Cleveland Clinic Hospital. Cost estimates reflect the duration of training by various supervisory RTs, their respective wages (including benefit costs), and educational materials used in training and orientation. RESULTS: Turnover rates ranged from 3% to 18% per year. Five of the 8 institutions from which rates were available reported rates greater than 8% per year. The rate of annual turnover correlated significantly with the ratio of hospital beds to RT staff (Pearson $r = 0.784$, $r^2 = 0.61$, $p = 0.02$). The cost of training an RT at the Cleveland Clinic Hospital totaled $3,447.11. CONCLUSIONS: Turnover among respiratory therapists poses a substantial problem because of its frequency and expense. Greater attention to issues affecting turnover and to enhancing retention of RTs is warranted. *Key words: employee turnover, employee retention, respiratory care, respiratory therapist, costs, training, trainee, staff, personnel, RT.* [Respir Care 2001;46(3):238–242]

Introduction

In all work settings, employee turnover is generally undesirable because services are disrupted and because training new employees incurs expense. In the specific context of respiratory care, respiratory therapists (RTs) leaving a department requires that new therapists undergo orientation and training, which requires time and associated expense. Furthermore, the momentum and camaraderie of a working clinical group is disrupted by the departure of veteran members.

In light of the adverse effects of turnover, reducing turnover is usually desirable, as it is in respiratory care. Yet, despite a widely shared goal of minimal turnover, neither the annual rate nor the associated expense of turnover of RTs has been described.

Indeed, little attention has been given to the general issue of RT recruitment and retention. In one of the few available related studies,[1–3] Matthews et al[1] described the reasons cited for RTs' leaving the field. The 3 most important determinants (in descending order of priority) were:

1. To seek more job satisfaction,
2. To seek higher salary, and
3. Because of lack of advancement opportunities.

Rawlins[2] later confirmed these reasons in a survey regarding attrition; a discriminant function analysis of predictor variables for leaving the field showed that issues of supervision, role stress, and goal stress were the most predictive factors.

In assessing the determinants of role stress, we hypothesized that work load, summarized for acute care hospitals

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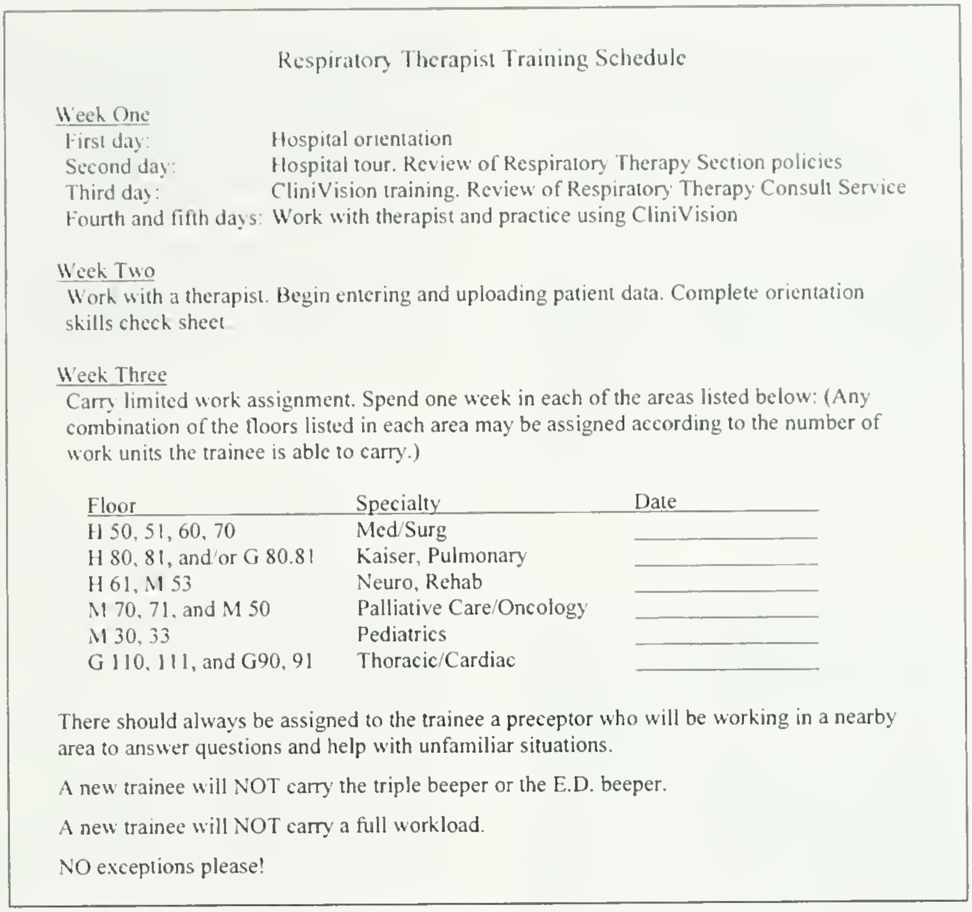

Respiratory Therapist Training Schedule

Week One

First day:	Hospital orientation
Second day:	Hospital tour. Review of Respiratory Therapy Section policies
Third day:	CliniVision training. Review of Respiratory Therapy Consult Service
Fourth and fifth days:	Work with therapist and practice using CliniVision

Week Two

Work with a therapist. Begin entering and uploading patient data. Complete orientation skills check sheet

Week Three

Carry limited work assignment. Spend one week in each of the areas listed below: (Any combination of the floors listed in each area may be assigned according to the number of work units the trainee is able to carry.)

Floor	Specialty	Date
H 50, 51, 60, 70	Med/Surg	
H 80, 81, and/or G 80.81	Kaiser, Pulmonary	
H 61, M 53	Neuro, Rehab	
M 70, 71, and M 50	Palliative Care/Oncology	
M 30, 33	Pediatrics	
G 110, 111, and G90, 91	Thoracic/Cardiac	

There should always be assigned to the trainee a preceptor who will be working in a nearby area to answer questions and help with unfamiliar situations.

A new trainee will NOT carry the triple beeper or the E.D. beeper.

A new trainee will NOT carry a full workload.

NO exceptions please!

Fig. 1. Schedule and activities in training a new respiratory therapist at the Cleveland Clinic Hospital.

as the ratio of hospital beds to RTs, would correlate with the rate of turnover among RTs. Thus, to address the gap in understanding determinants of RT retention, we undertook this descriptive study and asked RT representatives of the 9 participating hospitals in the Cleveland Clinic Health System to describe their hospitals' annual rates of RT turnover. In addition, for the Cleveland Clinic Hospital, we calculated the expenses associated with training an RT who is newly joining the staff.

Methods

The Cleveland Clinic Health System is composed of 9 participating hospitals that represent the full spectrum of hospital care, from smaller, private, community-based institutions to large, tertiary care institutions (Table 1). To elicit information about annual rates of turnover among RTs, we conducted a survey, both in person and by written questionnaire, of respiratory care supervisory personnel from each participating hospital. Specifically, we posed the question, "Over the last 3 years (October 1996 to October 1999), what is the annual rate at which RTs have left employment at your institution?" The survey was initiated at a system-wide meeting of representatives of all participating institutions in October 1999. Follow-up for questionnaire completion was finalized by February 2000. Responses were based on the attending RT leaders' reports, which were based on their consulting department records (but not official payroll or institutional human resource reports). For the sake of the survey, RTs were defined as all full-time employed therapists, irrespective of the level of credential (eg, registered respiratory therapist, certified respiratory therapy technician, on-the-job trainee).

To calculate the costs of training (Table 2) we reviewed the training schedule for an RT joining the Section of Respiratory Therapy at the Cleveland Clinic Hospital (Fig. 1). Cost estimates reflect the duration of training by various supervisory RTs, their respective wages (both direct and indirect), and an estimate of the equipment costs associated exclusively with respiratory care training (eg, policies, procedures, and handbooks). To reflect the expense of unfulfilled work by new trainees, the cost of the portion of a full work load that the new trainee is not yet able to assume is also included in the cost estimate.

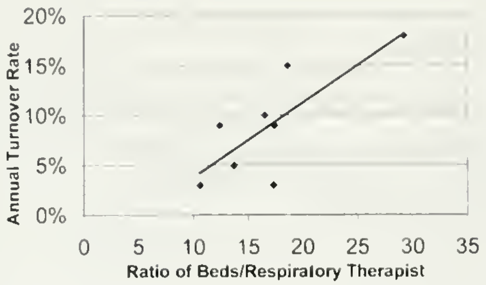

Fig. 2. Correlation between the rate of annual respiratory therapist (RT) turnover at 8 Cleveland Clinic Health System Hospitals and the ratio of hospital beds/RTs in those hospitals (Pearson r = 0.784, $r^2 = 0.61$, p = 0.02). The moderate but incomplete correlation suggests that the beds/RTs ratio (which we speculate reflects work load) is an important determinant of turnover but that other factors are also at play (eg, possibly salary, benefits, etc).

Results

Table 1 presents the rate of RT turnover (excluding as-needed staff) in participating hospitals. Notably, turnover rates ranged widely (3–18% per year), but 5 of the 8 institutions from which rates were available reported rates greater than 8% per year. The rate of annual turnover correlated significantly with the ratio of hospital beds/RTs (Fig. 2, Pearson r = 0.784, $r^2 = 0.61$, p = 0.02), suggesting that higher workloads may contribute to turnover of RTs. In particular, the hospital with the highest turnover rate (18% per year) also had the highest ratio of beds/RTs (29.2, see Table 1). In the same vein, one of the two hospitals with the lowest turnover rate (3% per year) had the lowest ratio of beds/RTs (10.6). However, the other hospital with low turnover had an intermediate ratio of beds/RTs (17.3), suggesting that offsetting features can encourage RT retention despite a high beds/RTs ratio or associated work load. Taken together, the moderate correlation (r = 0.784) between the annual turnover rate of RTs and the ratio of beds/RTs suggests that the ratio partially but incompletely explains turnover. Likely reasons for the modest degree of correlation include the presence of other determinants of turnover (eg, salary, benefits, and proximity of the hospital to the RTs' home) and the fact that the ratio of beds/RTs is an incomplete measure of work load.

Table 2 presents the costs of training an RT new employee at The Cleveland Clinic Foundation, which total $3,447.11. Institutions training several new RTs per year will incur costs that are multiples of this number. As an example, a hospital with 3 RTs leaving per year would incur a 5-year turnover cost of $51,707 (1999 United States dollars).

Discussion

In this study of the Cleveland Clinic Health System hospitals, which represent both community-based and tertiary care academic hospitals, the main findings regarding RT turnover are:

1. Annual rates of turnover are reasonably high, exceeding 8% per year in 5 of the 8 hospitals from which rates were available.
2. The estimated cost of training a new RT is high ($3,447.11 per therapist), suggesting that the aggregate cost of turnover can be substantial.
3. The rate of turnover correlated with the ratio of hospital beds/RTs, suggesting that greater coverage demands (and presumably greater workloads) contribute to turnover.

Taken together, these observations underscore the adverse effects of respiratory therapist turnover and recommend efforts to enhance retention in order to ameliorate these effects. Indeed, in addition to incurring substantial cost, turnover causes disruption of clinical care, the need to divert personnel from providing clinical care to providing training, and erodes both morale and the continuity of the clinical mission.

Table 1. Respiratory Therapist Turnover Rates over the Past 3 Years in Cleveland Clinic Health System Hospitals

Hospital	Number of Beds	Number of RT Staff	Ratio of Beds to RTs	Annual Turnover Rate Averaged Over 3 Years (%)	Average Number of Staff Leaving per Year
Lutheran	204	12	17.3	3	0.3
Marymount	279	32	8.7	NA	NA
Hillcrest	360	34	10.6	3	1.3
South Pointe	364	22	16.5	10	2.3
Euclid	371	20	18.6	15	3.0
Huron Road	379	13	29.2	18	2.3
Lakewood	400	23	17.4	9	2.0
Fairview	478	35	13.7	5	1.7
Cleveland Clinic	1,192	96	12.4	9	8.2

RT = respiratory therapist
NA = not available

Table 2 Cost of Training a Therapist at the Cleveland Clinic Hospital

Day	Activity	Personnel	Wages ($)
1	General hospital orientation	RT in-training	188.48
2	Hospital tour and section policies	RT in-training, Supervisor, and Education Coordinator	431.04
3	CliniVision training	RT-in-training and Education Coordinator	431.04
4–8	Training with an RT	RT-in-training's wages × 1/3 + RT's wages × 5	1,523.39
9–11	RT-in-training takes 1/4 workload	3/4 RT-in-training's wages × 3	424.08
12–14	RT-in-training takes 1/2 workload	1/2 RT-in-training's wages × 3	282.72
15–17	RT-in-training takes 3/4 workload	1/4 RT-in-training's wages × 3	141.36
	Printed education materials	—	25.00
		Total Costs	3,447.11

RT = respiratory therapist

Although this study is, to our knowledge, the first to examine the cost of turnover for RTs, our cost estimates are consistent with previous estimates for training other allied health care providers. Specifically, Bethel[4] reported a mean cost of $4,720.93 for orienting and training new nurses. Also assessing nurses, Giles and Moran[5] reported a cost range of $1,140 to $9,000 per nurse, depending on the extent of orientation. Clearly, like these earlier estimates for nurses, our cost estimates for training RTs depend on the specific elements of the training and the trainee's level of experience.[6] In this context, it is likely that the extent, duration, and cost of training would be lower for incoming RT employees who have extensive past experience than for new graduates.

Similarly, several features of respiratory care practice at the Cleveland Clinic Hospital may cause our estimates to exceed the training costs at other institutions lacking these features. Specifically, new RT training at the Cleveland Clinic includes orientation to the Respiratory Therapy Consult Service (a program by which respiratory care is allocated by RTs according to respiratory protocols) and training with a management information system (CliniVision, Mallinckrodt-Nellcor-Puritan Bennett, St Louis, Missouri) by which RTs receive work assignments and document services provided. Furthermore, orientation to a large institution such as the Cleveland Clinic Hospital (with 1,192 beds, Table 1) may be more time-consuming, complicated, and therefore costly than orientation to a smaller hospital. Notwithstanding these special features of RT training at the Cleveland Clinic Hospital that may overestimate training costs at other institutions, training is universally expensive. It should be noted that although as-needed and part-time staff were not included in these turnover rates, orienting staff that are not full-time employees incurs the same costs as employees who work full time. In fact, the costs may be higher in situations in which the part-time trainee is not available for orientation on a consecutively scheduled time period. Interruptions in the orientation schedule frequently cause more lengthy orientation time. Therefore, enthusiasm for retaining RTs also applies to as-needed staff.

The real impact of these findings is to focus attention on methods to optimize retention of RTs. Although this issue has received little formal attention in the literature to date, we believe that measures to enhance retention include creating a work environment in which RTs feel valued, capable of impacting clinical care, and empowered to use their clinical skills broadly in providing high quality respiratory care. Furthermore, though not addressed in this analysis, we speculate that implementing a respiratory care consult service confers several of these benefits. Specifically, in addition to enhancing the allocation of respiratory care services, use of a respiratory care consult service allows RTs to apply their assessment skills in generating a care plan under the guidance of a structured program. Not surprisingly, to the extent that respiratory care departments with protocol services enjoy institutional respectability and support, RTs in such departments are likely to feel valued. In possible support of this conjecture, we have observed a temporal association between lower rates of RT turnover and implementation of the Respiratory Therapy Consult Service at the Cleveland Clinic Hospital; that is, turnover decreased from 22.9% before the service was offered (1987) to 10.5% (1994) after implementation.

The cost of training new employees, although substantial, is only one of the disadvantages of employee turnover. Members of the health care team other than RTs (eg, physicians, nurses, and unit secretaries) are affected by the loss of an RT experienced in the routines and subtleties of a specific work area. Teamwork among the RTs and the flow of work between shifts can easily be disrupted when an experienced therapist leaves and a new employee is in the learning process. Additionally, time beyond the orientation period is often required for a new employee to develop the necessary efficiency, expertise, and confidence needed to participate in research projects and educational activities such as journal club meetings and inservices for other departments.

This being the first study to systematically address RT turnover in a single health care system, several limitations of the study are noteworthy. First, the data on turnover rates are based on the attendees' reviews of departmental records and memory. Certainly, though it is conceivable that official payroll or human resource department data may have differed, we suspect that these records by RT department leaders are accurate.

As mentioned above, a second limitation is that the cost estimates based on the one institution from which accurate estimates were available (The Cleveland Clinic Hospital) may not generalize to other settings.

Third, it must be remembered that correlation does not imply causality, so the observed relationship between the beds/RTs ratio and turnover does not establish it as a determinant of turnover. Notably, unlike the few available reports regarding RT attrition,[1–3] our study did not directly survey staff therapists. Nonetheless, our findings support the observation that role stress is an important determinant of RTs' desires to leave their positions.

In summary, our analysis shows that turnover among RTs is an important problem because of its frequency and expense. Our findings recommend efforts to enhance retention of RTs.

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Assessment of Aspiration in Patients with Tracheostomies: Comparison of the Bedside Colored Dye Assessment with Videofluoroscopic Examination

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BACKGROUND: Aspiration is a serious clinical concern in patients with long-term artificial airways. The purpose of this study was to determine the reliability of a bedside colored dye assessment of aspiration in tracheostomized patients and to determine its comparability to a more sophisticated videofluoroscopic study. METHODS: This was a prospective, blinded comparison study conducted in a large, urban, university teaching hospital. We studied 20 consecutive patients who underwent tracheostomy for bronchial hygiene needs and who were referred for videofluorographic evaluation for suspected oropharyngeal dysphagia and possible aspiration. Excluded were patients unable to follow verbal commands and those requiring mechanical ventilatory support. All patients were brought to the videofluorography suite for colored dye assessment for aspiration and videofluorographic assessment of oropharyngeal swallow. A nurse, blinded to the results of videofluorographic swallow study, performed colored dye assessments for aspiration. Speech-language pathologists, blinded to the results of the colored dye assessments, interpreted simultaneous (preliminary) and subsequent complete (final) videofluorographic evaluations of swallow. RESULTS: The colored dye aspiration assessments and the videofluoroscopic studies were compared for the frequency of aspiration detection. Sensitivity and specificity were determined using standard methods. Seven patients showed no aspiration on either the colored dye test or videofluoroscopic examination. Eight patients were judged to aspirate by videofluorography but not by the colored dye test. Five patients were judged to aspirate by both the colored dye test and videofluorography. The data indicate that the colored dye test for aspiration carries a low sensitivity of 38% (95% confidence interval = ± 7%), but a high specificity of 100%. The videofluoroscopic study detected a significantly greater frequency of aspiration than did the colored dye test ($p < 0.01$). CONCLUSIONS: The colored dye test for aspiration can provide useful information when positive, but because there is a significant false negative rate, decisions made on the basis of a negative test must be made with caution. *Key words: aspiration, colored dye assessment, videofluoroscopy, tracheostomy, swallow study.* [Respir Care 2001;46(3):243–247]

Background

Aspiration is a serious problem in patients with various disease processes. Although one of the accepted indications for placement of an artificial airway is protection of the airway from aspiration,[1] this subject is rather controversial. Additionally, oral feeding can be problematic in this patient population. Tracheopulmonary aspiration on oral feeding has been documented in tracheostomized patients with respiratory failure, head-neck cancer, or neu-

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rologic injuries.[2,3] Patients who require prolonged endotracheal intubation or tracheostomy have also been noted to have multiple defects in swallowing mechanism,[4] which heightens the importance of the question of the safety of oral feedings in patients who require prolonged use of artificial airways (ie, tracheostomy).

Sophisticated evaluation of swallow and airway protection mechanisms involves videofluoroscopic, flexible fiberoptic endoscopic, and scintigraphic techniques. These methods may require expensive equipment, costly personnel time, and exposure of the patient to ionizing radiation, and often present transport difficulties. Various means of bedside evaluation for aspiration exist.[5,6] One of the simplest and most cost-effective methods is to deflate the cuff of the tracheostomy tube and allow the patient to swallow a small volume of liquid containing a nontoxic food dye. Following the swallow procedure, tracheal secretions are suctioned and inspected for the presence of the colored dye. If the tracheal secretions are pigmented by the dye, aspiration has occurred. Conversely, the absence of pigment implies no aspiration has occurred.[7] The sensitivity of this method of testing has been questioned by Thompson-Henry and Braddock.[8,9] However, the validity of the scientific methods used by Thompson-Henry and Braddock[8] has been challenged because the patients studied were neither consecutive nor randomized and the comparative methods of aspiration assessment were not performed concurrently.[10,11] The purpose of our study was to compare a bedside colored dye assessment of aspiration, as it is performed clinically, with a more sophisticated videofluoroscopic study, to assess the reliability of the bedside method in tracheostomized patients.

Materials and Methods

Approval for this study was obtained from the Institutional Review Board of Northwestern University Medical School and the Northwestern Memorial Hospital Research Committee. Informed consent was obtained from all patients. Patients were placed in the upright position in the fluoroscopy equipment (Serigraph, Siemens Medical System, Hoffman Estates, Illinois) so that a lateral view of the oral cavity and pharynx could be observed. The fluoroscopic tube was focused on the lips anteriorly, the pharyngeal wall posteriorly, the soft palate superiorly and the cervical esophagus inferiorly. The tracheostomy tube cuff was then deflated, and the patient was presented with approximately 100 mL of a solution consisting of 50% water, 50% liquid barium (Sol-O-Pake barium sulfate suspension, EZEM, Westbury, New York), and 5 drops of a standard commercially available nontoxic blue food dye (Blue #1). The patient was presented with the radio-opaque, colored solution via straw, cup, or syringe, depending on his or her ability to accept various utensils into the mouth. The volume of liquid taken by the patient was not controlled except when liquid was administered via syringe, in which case the volume was restricted to 10 mL (2 teaspoons). Patients were then instructed to swallow, and videofluoroscopy was begun. Since the goal of the study was to compare the reliability of the bedside test with that of more sophisticated testing regimens, efforts were directed at duplicating the relatively uncontrolled methods of clinical assessment regularly performed by various health care professionals (eg, nursing, speech-language pathologists, and respiratory care practitioners). The procedures were recorded on videotape. Suctioning was performed with a standard 14 French suction catheter (Smiths Industries Medical Systems, Keene, New Hampshire) (4.6 mm in diameter and 30.5 cm long) immediately following the swallow attempt. The tracheobronchial secretions were inspected for the presence of dye after the initial swallow attempt. A manual resuscitation device (Laerdal silicone resuscitation bag, Laerdal Medical, Armonk, New York) was used to inflate the patient's lungs with an oxygen-enriched gas to prevent hypoxemia during suctioning, as is normally done clinically. During this initial swallow maneuver the patient was also observed for radiographic evidence of aspiration (preliminary videofluoroscopic study). The presence or absence of aspiration was indicated by (1) the presence or absence of colored dye in the secretions suctioned from the airways, and by (2) the simultaneous videofluoroscopic recording, and was noted for later data analysis. There were no delayed (ie, 24–48 h) assessments for aspiration of colored materials performed beyond the assessment made in the study session.

Immediately following the colored dye and initial videofluoroscopic assessment, a clinically indicated, complete modified barium swallow study (final videofluoroscopic study) was performed on each patient. The patient remained seated and was viewed in the lateral plane for the remainder of the study. The videofluoroscopic study began with two swallows of 1 mL of liquid barium and progressed to two swallows of 3 mL, 5 mL, and 10 mL, as tolerated. If aspiration occurred with larger volumes, standard interventions defined by speech-language pathology protocols were used in an attempt to eliminate the aspiration. Following administration of the liquid volumes, two swallows of 1 mL of barium pudding and two swallows of one quarter of a cookie (Lorna Doone, Nabisco Foods, East Hanover, New Jersey) coated with barium paste were presented. Aspiration events were noted for later data analysis. The results of the videofluoroscopic studies were considered positive if aspiration was detected on either the preliminary or final videofluoroscopic study. *Aspiration* on videofluoroscopy was defined as material entering the airway below the level of the true vocal folds. *Penetration* was defined as material entering the larynx, but remaining above the superior surface of the true vocal folds.[12] Penetration was not considered as an outcome in this study.

Results from the colored dye aspiration assessments and the videofluoroscopic studies were compared for the frequency of aspiration detection with McNamara's test.[13] Statistical significance was set at $p < 0.05$. Sensitivity and specificity were determined using standard methods.[14]

Results

Table 1 shows data relating to patient demographics, underlying disease processes, duration of tracheostomy, and results of aspiration assessments. Seven patients showed no aspiration on either the colored dye test or the videofluorographic examination. Eight patients were judged to aspirate by videofluorography but not by the colored dye test. Two of these patients (Table 1, #15 and # 19) did not show aspiration on either the colored dye test or the preliminary videofluorographic assessment but aspiration events were detected with materials of different consistencies during the subsequent full videofluorographic swallow assessments. Five patients were judged to aspirate by both the colored dye test and videofluorography. If the videofluoroscopic study is considered the accepted standard for the evaluation of aspiration, these data would indicate that there were zero false positive, 8 false negative, 5 true positive, and 7 true negative results obtained with the colored dye test (Fig. 1). On this basis, the colored dye test for aspiration carries a specificity of 100% but a sensitivity of only 38% (95% confidence interval = ± 7%). If the two patients who did not aspirate on the colored dye test or the preliminary videofluorographic study are considered to be true negatives, then the sensitivity becomes 45% (95% confidence interval = ± 8%) and the specificity remains 100%. The positive predictive value of the colored dye test (ability to predict the presence of aspiration) is 100%, whereas the negative predictive value (ability to predict lack of aspiration) is 47% (± 8%). The overall diagnostic accuracy (ability to correctly predict presence or absence of aspiration) of the colored dye test is 60% (± 9%). The videofluorographic technique detected a significantly greater frequency of aspiration than did the colored dye test ($p < 0.05$).

Table 1. Patient Demographics and Test Results

Subject	Age (y)	Sex	Pathology	Duration of Tracheostomy (d)	Colored Dye Test	Preliminary Video-Fluoroscopic Study	Final Video-Fluoroscopic Study
1	37	M	SCI	10	−	−	−
2	66	M	MI	79	−	+	+
3	18	F	SCI	10	−	+	+
4	46	M	RF	6	−	−	−
5	82	M	CVA/RF	9	+	+	+
6	50	F	SCI	21	−	+	+
7	35	M	SCI	18	−	−	−
8	45	M	SCI	46	−	−	−
9	76	M	COPD	19	+	+	+
10	35	F	MEN	13	−	−	−
11	81	F	SCI	60	−	+	+
12	28	M	SCI	62	−	−	−
13	70	M	SCI	34	−	−	−
14	43	M	ENC	44	+	+	+
15	25	M	CHI	28	−	−	+
16	58	M	SCI	43	+	+	+
17	67	M	COPD	33	−	+	+
18	53	M	SCI	26	−	+	+
19	62	M	LCA	240	−	−	+
20	27	M	SCI	24	+	+	+
Mean ± SD	50 ± 19			91 ± 271			

SCI = spinal cord injury
MI = myocardial infarction
RF = respiratory failure
CVA = cerebrovascular accident
COPD = chronic obstructive pulmonary disease
MEN = meningitis
ENC = encephalitis
CHI = closed head injury
LCA = laryngeal cancer

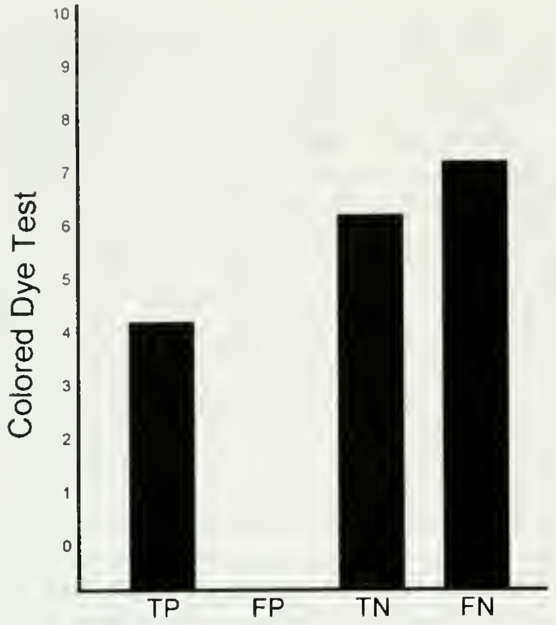

Fig. 1. Results of colored dye and videofluoroscopic swallow studies. TP = true positive. FP = false positive. TN = true negative. FN = false negative.

Discussion

Multiple defects in swallow mechanisms have been observed in patients who require prolonged endotracheal intubation or tracheostomy.[15] Tracheopulmonary aspiration of normal oropharyngeal secretions or during oral feeding in tracheostomized patients is a persistent problem that has been recognized for some time. Using a tracheostomy tube that has a high-volume, low-pressure cuff minimizes the aspiration of normal oropharyngeal secretions,[16] which is usually sufficient to prevent important complications, although low-level aspiration may still occur. The clinical consequences of such chronic, low-level aspiration are poorly defined at present and merit further study.

The issue of airway protection during oral feeding of tracheostomized patients is controversial. Following placement of a tracheostomy, clinical experience indicates that some would consider it inappropriate to allow oral feeding in virtually any patient; others orally feed tracheostomized patients with little or no hesitation and stop only if clinical problems are recognized. These two approaches each have important disadvantages. Restricting oral feeding in all tracheostomized patients unnecessarily denies many a degree of normalcy that may be psychologically critical to them. These patients already have substantial compromise of their lifestyles, and nonoral feeding only contributes further to that situation. In addition, there are serious potential complications associated with nonoral feedings, which, though infrequent, must be taken into consideration.[17,18] The alternative approach exposes patients to the risks of aspiration, including pneumonia, increased bronchial secretions (bronchorrhea), bronchospasm, hypoxemia, and more severe respiratory failure. The most prudent approach to that situation is to recognize that some tracheostomized patients will aspirate oral feedings, whereas others will not, and to make an assessment before such feedings are begun. There are various ways to conduct this evaluation. The most sophisticated include videofluoroscopic swallow studies, which permit the speech and language pathologist to (1) detect aspiration, (2) assess the physiologic or anatomic abnormalities that result in aspiration, and (3) define interventional strategies to stop the aspiration.[19–21] Unfortunately, some patients are not candidates for such studies because of respiratory, hemodynamic, or neurologic instability. Therefore, bedside techniques are frequently used to make determinations regarding the safety of oral feeding in those patients.

The original Evans blue dye test was used to detect ongoing aspiration, but not in conjunction with oral feeding.[5] This test entailed periodic (every 4 h) placement of several drops of Evans blue dye on the patient's tongue, followed by inspection of routinely suctioned respiratory secretions for the presence of the dye marker. The bedside techniques currently used to assess the safety of oral feeding include modifications of the original colored dye test, as outlined in the methods section. Recent case reports have documented that the standard colored dye aspiration assessment has a substantial false negative rate.[8] In light of this information and the frequency with which this test is used clinically, it was important to define the power and the limits of the colored dye test for aspiration. The data presented here indicate that the test is 100% specific for the detection of aspiration, but is of limited sensitivity (38–45% sensitivity, ± 7–8%, respectively).

Although this study demonstrates the weakness of the bedside assessment, there are study limitations that merit comment. The study sample population was small and rather heterogeneous. A larger and more focused patient population might provide more robust results. In addition, there was no attempt to improve the accuracy of the bedside testing system. These issues are the subjects of projects currently in progress.

These findings are important because they indicate a serious limitation of a commonly used bedside assessment. However, this does not obviate the fact that clinical decisions need to be made in an efficient and cost-effective manner. When used as a screening test, the bedside colored dye test for aspiration can provide useful information when positive, because of its high predictive value. However, decisions made on the basis of a negative test must be tempered with the understanding that the test carries a significant false negative rate. Clinical decisions (eg, regarding oral vs nonoral feeding) made based on false negative results may seriously impact the care and outcome of many acutely and chronically ill patients.

Conclusions

The care of these patients requires an integrated and multidisciplinary approach in order to be optimal and cost-effective. Cooperation and clear communication between physicians, nurses, respiratory therapists, and speech-language pathologists are essential to the development of an appropriate plan of care that encompasses all clinical concerns and considerations regarding tracheostomized patients. Bedside assessment of aspiration in tracheostomized patients is very useful if positive, but must be interpreted cautiously if negative. Videofluoroscopic evaluations of the swallow mechanism will detect the presence of aspiration more frequently than bedside techniques and will help guide therapeutic interventions.

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Heliox Delivery with Noninvasive Positive Pressure Ventilation: A Laboratory Study

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BACKGROUND: There is clinical interest in the use of heliox (helium-oxygen mixture) during noninvasive positive pressure ventilation (NPPV), but delivery of heliox with ventilators designed for NPPV has not been reported. We studied helium concentration ([He]) when an 80%:20% helium:oxygen mixture (heliox) was used with 5 NPPV ventilators (Knightstar, Quantum, BiPAP S/T-D30, Sullivan, and BiPAP Vision). METHODS: A simulated spontaneous breathing lung model was connected to the ventilators with a circuit incorporating a standard leak. Heliox flows of 0, 5, 10, and 18 L/min and oxygen flows of 0 and 10 L/min were titrated into the system at either a proximal position near the lung model or a distal position near the ventilator (titration method). Because the BiPAP Vision has an oxygen delivery module, it was also studied using heliox connected to the air inlet of an oxygen blender, with the blender outlet connected to the oxygen module of the ventilator (blender method). All ventilators were evaluated in spontaneous/timed mode at inspiratory/expiratory pressures of 10/5, 15/5, and 20/5 cm H_2O. After 5 minutes, [He], oxygen concentration, and pressure in the lung model were recorded. RESULTS: Heliox flow, NPPV settings, site of heliox infusion, and type of ventilator significantly ($p < 0.05$) affected [He]. [He] was > 60% when heliox flow was 18 L/min in some combinations of settings. The BiPAP S/T-D30 and Quantum occasionally functioned erratically. The BiPAP Vision (blender method) ventilator performed erratically with heliox unless the exhalation port test was bypassed on startup. The addition of heliox flow had no important effect on inspiratory or expiratory positive airway pressure on those breaths during which the ventilators functioned correctly. CONCLUSION: Heliox flow was the most important determinant of [He] when using heliox with NPPV. With heliox there was a potential for ventilator malfunction in some conditions. The clinical implications of these findings remain to be determined. *Key words: noninvasive positive pressure ventilation, heliox, mechanical ventilation, respiratory failure, helium.* [Respir Care 2001;46(3):248–254]

Background

In selected patients, noninvasive positive pressure ventilation (NPPV) decreases the need for endotracheal intubation and may offer a survival benefit, compared with conventional therapy or invasive ventilatory support.[1] The beneficial effects of NPPV for patients with acute respiratory failure secondary to chronic obstructive pulmonary disease (COPD) has been established in well conducted studies.[2] However, the use of NPPV for acute asthma has not been well studied and reports of its use in that setting are anecdotal.[3]

Heliox is a mixture of helium and oxygen. Heliox was first used by Barach in 1934 for patients with diseases characterized by partial airway obstruction.[4] Because it is less dense than air and oxygen, heliox may improve gas flow through partially obstructed airways. Case series and randomized trials of heliox in acute asthma have reported benefit.[5–10] Heliox has been reported as beneficial for invasively ventilated asthma patients,[5] and benefit has also been reported with the combined use of NPPV and heliox in COPD patients.[11–14]

NPPV is often administered using a portable pressure ventilator designed for that purpose.[1] Supplemental oxy-

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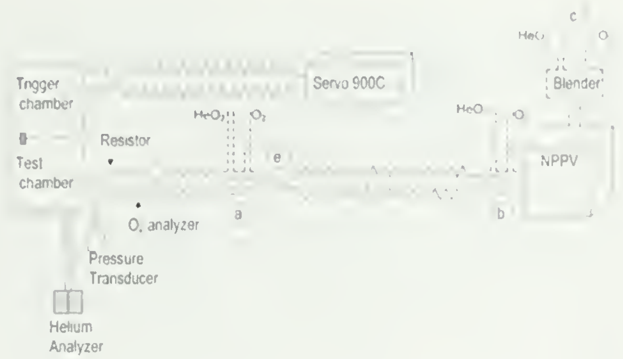

Fig. 1. Experimental setup (solid lines) and sites where heliox and oxygen were infused (dashed lines) into the Knightstar, Quantum, BiPAP S/T-D30, and Sullivan, and in the titration method with the BiPAP Vision. (a) proximal position, (b) distal position, (c) heliox and oxygen mixed with a blender before connected to the BiPAP Vision (blender method), and (e) the exhalation port (whisper swivel). NPPV = noninvasive positive-pressure ventilator.

gen with those ventilators is provided by titration into the mask or circuit near the patient or into the circuit at the ventilator outlet.[15] Because these ventilators are blower devices (in other words, they create pressure by blowing room air into the circuit), it is difficult to achieve a high oxygen concentration when oxygen is titrated into the system. Recently, there has been interest in administration of heliox during NPPV using a similar approach. This raises several questions. First, what helium concentrations can be achieved with titration of heliox into the circuit? Second, does titration of heliox into the ventilator circuit affect performance of the ventilator? We designed this study to address these questions. We hypothesized that it would be difficult to achieve high helium concentrations by titration of heliox into the circuit.

Methods

Bench Model

A two-chambered lung model (Michigan Instruments model 1600 Adult TTL, Michigan Instruments, Grand Rapids, Michigan) was used to simulate spontaneous breathing (Fig. 1). A Servo 900C ventilator (Siemens, Danvers, Massachusetts) was attached to one chamber (trigger chamber). The ventilator attached to the trigger chamber was set to a respiratory rate of 20 breaths/min. A small metal bar was placed between the chambers of the lung model such that inflation of the trigger chamber resulted in triggering of the ventilator attached to the contralateral chamber (test chamber). The lift bar was attached to the trigger chamber, but the test chamber was free to rise independently of the trigger chamber once the test breath was initiated. Sufficient positive end-expiratory pressure was applied to the trigger chamber to prevent separation of the two chambers at end-expiration. The test chamber compliance was set at 0.05 L/cm H_2O, and a resistance of 5 cm H_2O/L/s was used (linear resister model #7100, Hans Rudolph, Kansas City, Missouri). Using a super-syringe (Hamilton, Reno, Nevada), the compliance of the lung model was found to be 0.0488 L/cm H_2O. The lung model settings were chosen to assure the target tidal volumes of 250, 500, and 750 mL at BiPAP settings of 10/5 cm H_2O, 15/5 cm H_2O, and 20/5 cm H_2O, respectively. A short tube of 100 mL was inserted between the resister and the ventilator circuit.

Tested Ventilators

Five noninvasive positive pressure ventilators were studied: BiPAP S/T-D30 (Respironics, Murrysville, Pennsylvania), Knightstar 335 (Mallinckrodt, Pleasanton, California), Quantum PSV (Respironics/Healthdyne Technologies, Marietta, Georgia), Sullivan VPAP II ST (ResMed, San Diego, California), and BiPAP Vision (Respironics, Murrysville, Pennsylvania). For the Knightstar 335, inspiratory sensitivity was set at 3 and expiratory sensitivity was set at 2. Rise time was set at 0.1 s for the Quantum PSV and BiPAP Vision. For the Sullivan VPAP II ST, rise time was set at minimum (< 300 ms) and IPAP max was set at 1.8 s. All ventilators were set in spontaneous/time (S/T) mode with a backup rate of 10/min. Thus, inspiration was always triggered by the lung model. The test chamber was connected to the tested ventilator with a standard BiPAP disposable circuit (#312107, Respironics, Murrysville, Pennsylvania) equipped with a Whisper Swivel (Respironics, Murrysville, Pennsylvania).

Study Protocol

Heliox was supplied as an 80%:20% helium:oxygen mixture in a high-pressure cylinder (BOC Gases, Hingham, Massachusetts). Cylinder pressure was reduced to 50 psig (Western Medica regulator, Westlake, Ohio). An oxygen flow meter (Precision Medical, Northampton, Pennsylvania) was attached to the pressure regulator. This flow meter was adjusted to settings of 0, 3, 6, and 10 L/min to deliver heliox flows of 0, 5, 10, and 18 L/min, respectively. The heliox flow from the oxygen flow meter was confirmed by timed collection of heliox into a weather balloon (Vacumed, Ventura, California) and measurement of the volume with the calibrated super syringe.

Heliox flow was added at either a proximal position (between the simulated trachea and the exhalation port) or a distal position (at the ventilator outlet) (see Fig. 1). Using a second oxygen flow meter (Precision Medical, Northampton, Pennsylvania), oxygen flows of 0 and 10 L/min were also added at the same sites as heliox. Inspiratory/expiratory pressure settings of 10/5, 15/5, and 20/5 cm H_2O were set on the tested ventilators.

The BiPAP Vision differs from the other ventilators in that it has an oxygen module that allows the desired fraction of inspired oxygen (F_{IO_2}) to be set. Heliox delivery with this ventilator was evaluated using two approaches. First, the heliox titration method at the proximal and distal sites was used as described above, with F_{IO_2} settings of 0.21 and 0.40. Second, heliox was delivered directly into the oxygen module using an external oxygen blender (Bird/3M, Palm Springs, California). The 80%:20% helium:oxygen mixture at 50 psig was connected to the air inlet of an oxygen blender, and oxygen was added to the oxygen inlet of the blender in the usual manner. The outlet of the blender was connected to the oxygen module of the ventilator. The F_{IO_2} on the BiPAP Vision was fixed at 1.0 to deliver only the gas mixture from the external blender. The F_{IO_2} was adjusted on the blender for 0.21, 0.3, 0.4, 0.5, 0.6, 0.7, 0.8, 0.9, and 1.0. For the blender method, the performance of the ventilator was evaluated either with or without performing the exhaust port test at ventilator startup.

Measurements and Calibration

After 5 minutes of each combination of settings, helium concentration ([He]) and oxygen concentration ([O_2]) in the test lung bellows were measured using a helium analyzer (PK Morgan, Chatham, Kent, England) and an oxygen analyzer (oxygen monitor 7820, Mallinckrodt, Pleasanton, California). Pilot studies showed no difference in [He] at 5 minutes and 15 minutes. The helium analyzer was calibrated with heliox (80%:20% helium:oxygen). Because of the measurement range of the helium analyzer, the gas mixture to be analyzed was diluted with a super syringe to a 1:5 ratio. The oxygen analyzer was calibrated with room air and 100% oxygen.

Table 1. Average Helium Concentration

Ventilator	IPAP/EPAP Setting (cm H_2O)	Heliox Flow (L/min)		
		5	10	18
Knightstar	10/5	29 ± 15	51 ± 14	71 ± 7
	15/5	20 ± 4	37 ± 4	60 ± 1
	20/5	18 ± 3	35 ± 6	57 ± 3
Quantum	10/5	31 ± 18	48 ± 18	69 ± 8
	15/5	19 ± 6	38 ± 5	59 ± 6
	20/5	18 ± 4	36 ± 5	59 ± 4
S/T-D 30	10/5	22 ± 12	38 ± 13	58 ± 14
	15/5	18 ± 5	37 ± 5	54 ± 12
	20/5	18 ± 4	37 ± 4	54 ± 5
Sullivan	10/5	26 ± 13	44 ± 10	65 ± 5
	15/5	19 ± 3	37 ± 2	63 ± 7
	20/5	19 ± 2	37 ± 3	60 ± 7
Vision	10/5	23 ± 9	42 ± 12	64 ± 12
	15/5	16 ± 4	34 ± 7	56 ± 9
	20/5	15 ± 3	31 ± 5	56 ± 7

Data are mean ± SD. Data are an average of values from the proximal and distal positions.
IPAP = inspiratory positive airway pressure
EPAP = expiratory positive airway pressure

Airway pressure at the test lung bellows was measured using a pressure transducer (MP 45–32-871, ± 100 cm H_2O, Validyne, Northridge, California). The pressure transducer was calibrated at 20 cm H_2O with a water manometer. The airway pressure was continuously recorded for at least 6 breaths. All signals were amplified (Hewlett Packard model 8805C, Waltham, Massachusetts), digitized at 100 Hz, and recorded using WINDAQ (Dataq Instruments, Akron, Ohio). WINDAQ playback software was used to analyze the data. Inspiratory and expiratory pressures for all settings were analyzed. Any abnormal pattern, either triggering or cycling, that occurred during the evaluation period was defined as erratic performance.

Statistical Analysis

Data are presented as mean ± SD. Comparisons were done using univariate analysis of variance. When F ratios showed significant differences, the Scheffé test was used for post hoc analysis. Differences were considered significant at $p < 0.05$. All statistical analysis was performed using commercially available software (SPSS 9.0, SPSS, Chicago, Illinois).

Results

Helium Concentration

Table 1 shows the effect of different heliox flows at different ventilator settings. [He] was significantly affected by heliox flow ($p < 0.001$), NPPV settings ($p < 0.001$), site of heliox infusion ($p < 0.001$), and ventilator type ($p < 0.05$). [He] was not affected by oxygen flow ($p = 0.063$). [He] was more than 60% when heliox flow was 18 L/min at some combinations of settings. By post hoc analysis, the [He] at an NPPV setting of 10/5 cm H_2O was significantly ($p < 0.001$) greater than the settings of 15/5 cm H_2O and 20/5 cm H_2O. [He] from the Knightstar was significantly ($p = 0.049$) different than that from the BiPAP S/T-D 30, but not different from the Quantum ($p = 1.000$) and Sullivan ($p = 0.963$). Figure 2 shows the helium concentrations when heliox was infused into the system at the different positions.

When heliox was delivered by the titration method with the BiPAP Vision, [He] was significantly affected by heliox flow ($p < 0.001$), NPPV settings ($p < 0.05$), and site of heliox infusion ($p < 0.001$). [He] was not affected by F_{IO_2} setting on the Vision ($p = 0.753$). [He] was more than 60% when heliox flow was 18 L/min (see Table 1). When

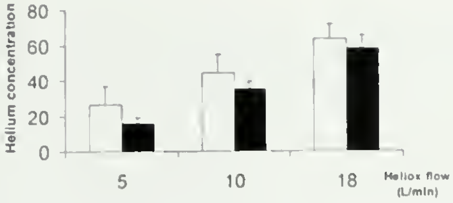

Fig. 2. Helium concentration when heliox was infused into the Knightstar, Quantum, BiPAP S/T-D30, and Sullivan at the proximal position (open bars) and at the distal position (closed bars) with heliox flows of 5, 10, and 18 L/min. $p < 0.001$ for differences in site of infusion and $p < 0.001$ for heliox flow.

heliox was delivered with the blender and the BiPAP Vision, [He] was significantly ($p < 0.001$) affected by the blender setting (Fig. 3), but was not affected by NPPV settings ($p = 0.202$).

Performance of the Ventilators

The ventilators occasionally performed erratically when heliox flow was added to the system (Table 2 and Fig. 4). The BiPAP S/T-D 30 performed erratically at 3 of the 48 settings tested. The Quantum performed erratically at one of the 48 settings tested. The Knightstar, Sullivan, and BiPAP Vision (titration method) did not function erratically at any tested settings. It was observed that the Knightstar had a greater trigger delay with a higher heliox flow into the system (Fig. 5). With heliox delivery by blender and the BiPAP Vision, the ventilator triggered and cycled erratically at 8 of the 27 settings tested. However, the BiPAP Vision functioned correctly at all evaluated settings when the exhaust port test was bypassed at the ventilator startup. On those breaths where the ventilators functioned appropriately, the inspiratory and expiratory pressures were significantly ($p < 0.001$) affected by heliox flow. However, the maximum IPAP difference was only 0.52 cm H_2O when comparing 18 L/min to no heliox flow. Similarly, the maximum end-positive airway pressure difference was only 0.22 cm H_2O.

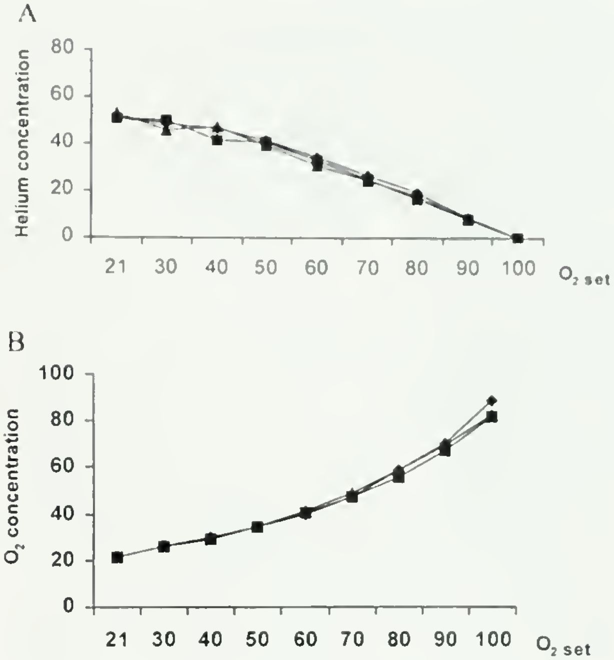

Fig. 3. A: Helium concentration with the BiPAP Vision when helium-oxygen mixture was adjusted with the external blender between 21% and 100% oxygen settings at 10/5 cm H_2O (♦), 15/5 cm H_2O (■), and 20/5 cm H_2O (▲). B: Oxygen concentration when helium-oxygen mixture was adjusted with the external blender between 21% and 100% oxygen settings at 10/5 cm H_2O (♦), 15/5 cm H_2O (■), and 20/5 cm H_2O (▲).

Table 2. Settings of Erratic Performance of Each Ventilator

Ventilator	IPAP/EPAP Setting (cm H_2O)	HeO_2 Flow (L/min)	O_2 Flow (L/min)	Position of Infusion
Quantum	20/5	18	10	Distal
BiPAP S/T-D30	10/5	10	0	Proximal
	20/5	5	0	Distal
	20/5	5	10	Distal
BiPAP Vision	10/5	21	—	Blender
	15/5	21	—	Blender
	15/5	60	—	Blender
	20/5	30	—	Blender
	20/5	40	—	Blender
	20/5	50	—	Blender
	20/5	60	—	Blender
	20/5	70	—	Blender

IPAP = inspiratory positive airway pressure
EPAP = expiratory positive airway pressure
For the BiPAP Vision, HeO_2 flow indicates the fraction of inspired oxygen setting on the external blender

Discussion

The principal findings of our study are as follows.

1. Using a portable noninvasive positive pressure ventilator, helium concentrations of about 60% can be delivered.
2. Heliox flow is the major determinant of [He].
3. Heliox does not have an important effect on the inspiratory and expiratory pressure settings.
4. Heliox flow may occasionally cause erratic triggering and cycling of some ventilators.

The factor that had the greatest effect on [He] was the flow of heliox titrated into the ventilator circuit. Another important determinant of [He] was the ventilator settings. A higher [He] was delivered with a lower IPAP setting, presumably because this resulted in a lower tidal volume

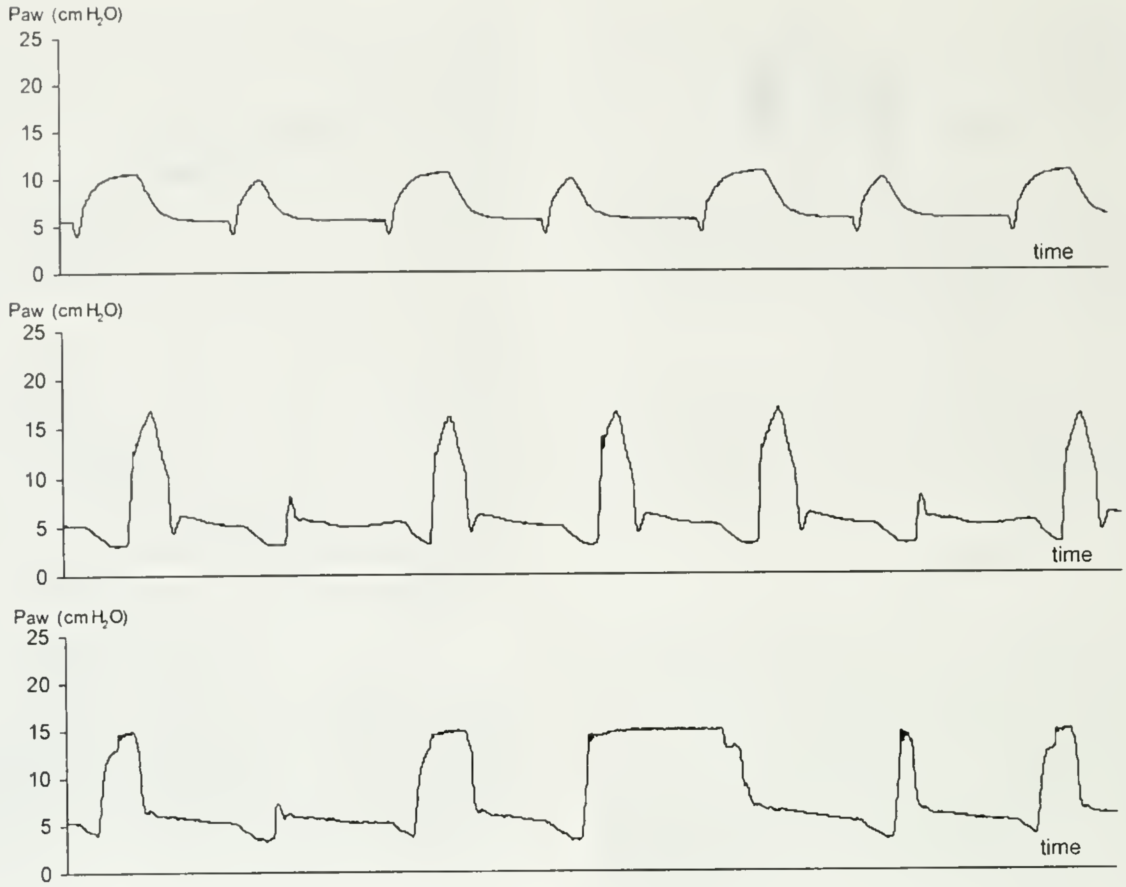

Fig. 4. Airway pressure (P_{aw}) waveforms showing erratic performance of: (upper panel) the BiPAP S/T-D30 when heliox flow of 10 L/min was titrated into the system at proximal position with noninvasive positive-pressure ventilation (NPPV) set at 10/5 cm H_2O; (middle panel) the Quantum when heliox flow of 18 L/min was titrated into the system at distal position with NPPV set at 20/5 cm H_2O; and (lower panel) the BiPAP Vision when heliox mixture was delivered with external blender and adjusted at 21% oxygen setting with NPPV set at 15/5 cm H_2O.

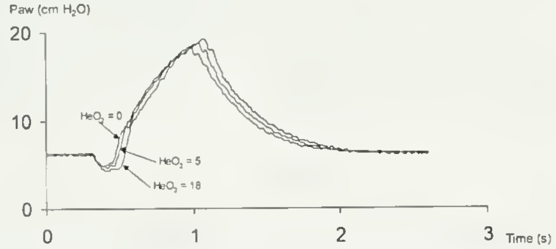

Fig. 5. Effect of heliox (HeO_2) flow on the trigger sensitivity of the Knightstar with titration into the system at the distal position and noninvasive positive-pressure ventilation (NPPV) setting was 20/5 cm H_2O. P_{aw} = airway pressure.

and thus a higher [He] for a fixed heliox flow. Although the site of heliox infusion and ventilator type affected the [He], these effects are probably too small to be considered clinically important. However, for a given ventilator, our data suggest that the proximal infusion site should be used to maximize the delivered [He].

Within the past decade, there have been a growing number of randomized, controlled trials reporting the effectiveness of noninvasive ventilation.[1] In COPD patients, Jaber et al[14] reported benefit with NPPV and heliox delivered via a prototype ventilator. Jolliet et al,[13] using a critical care ventilator, also reported benefit with heliox and NPPV for COPD. In neither of those studies did the investigators use portable pressure ventilators that are commonly used to provide NPPV. To our knowledge, the performance of portable pressure ventilators with heliox has not been previously reported.

The higher the helium concentration of the inspired gas, the greater the likelihood of benefit. Although the lowest effective helium concentration is not known, physiologic effects of heliox have been reported with helium concentrations as low as 50% in patients with large airway obstruction.[16–19] In COPD patients, Jolliet et al[13] fixed the helium concentration at 70%. Also in COPD patients, Jaber et al[14] reported F_{IO_2} ranging from 0.25 to 0.40, implying that helium concentrations of 60% to 75% were used. Our data indicate that helium concentrations of > 60% can be delivered with some portable pressure ventilators if a high heliox flow is used (eg, 18 L/min). Because ours was a bench evaluation, we do not know the clinical benefit, if any, of heliox delivery with portable pressure ventilators.

There is a potential for ventilator malfunction whenever heliox is used with ventilators that have been calibrated to function with air or oxygen.[20] Portable pressure ventilators for noninvasive ventilation are designed to operate in the presence of a fixed leak. Because of the lower density of heliox, gas flow is greater through this fixed leak. We were concerned that this might affect the ability of the ventilator to trigger and cycle effectively. For the Knightstar ventilator, the trigger was affected slightly by the addition of heliox (see Fig. 5). We also found that the addition of heliox caused issues with erratic performance for some ventilators at a few settings (see Table 2 and Fig. 4). These findings many not be clinically important, because the heliox effect on triggering for the Knightstar was small and the erratic performance of the S/T-D30 and the Quantum occurred only at a few of the many settings that we tested. There was no apparent effect of heliox on the function of the Sullivan VPAP. Because helium does not affect the performance of pressure transducers, we were not surprised that the IPAP and end-positive airway pressure settings were not affected by heliox to any important degree on any of the tested ventilators. Although statistically significant, the effects of heliox on inspiratory and expiratory pressures were too small to be considered clinically important and are within the error of measurement.

The Vision ventilator allows the user to conduct an exhalation port leak test at the time of ventilator startup, which is intended to improve triggering accuracy. When heliox is delivered through the Vision ventilator (external blender), the differences in gas density between the exhalation port test and subsequent heliox delivery results in erratic triggering and cycling of the ventilator (Peter Hill PhD, Respironics, Murrysville, Pennsylvania, 2000, personal communication). When heliox was delivered after bypassing the exhalation port leak test at start-up, we found that the ventilator functioned correctly. It is ironic that a feature of this ventilator designed to improve triggering actually worsened triggering when heliox was used. In fact, the ventilator functioned more correctly with heliox when this feature was bypassed. It is also interesting to note that the [He] delivered was only about 60% when the Vision ventilator was set to 100%—a setting that theoretically should deliver 80% helium. The reasons for this are not entirely clear. However, it is important to note that the Vision, like all portable noninvasive ventilators, is a blower device. As such, gas from the oxygen module is diluted with room air. This is illustrated by the delivered oxygen concentrations from this ventilator (see Fig. 3B). We suspect that this effect may be magnified when heliox is introduced to the oxygen module of this ventilator.

It is important to appreciate the effect of heliox titration with a noninvasive ventilator on resource utilization. A typical heliox cylinder contains 4,500 L of gas. At a flow of 18 L/min, the duration of flow from this cylinder is about 4 hours. In other words, 6 gas cylinders, at a cost of $85/cylinder, are required for 24 hours of therapy (about $500 for one day of therapy). Accordingly, NPPV with heliox should be regarded as short-term therapy as definitive therapy is initiated.

It may have been possible to achieve higher helium concentrations if we used 100% helium rather than 80% helium as our source gas. However, we believe that that approach is dangerous because it permits the possibility of delivering a hypoxic gas mixture. Accordingly, we strongly discourage using 100% helium as the source gas for heliox therapy. We might have been able to achieve higher helium concentrations with higher heliox flows. We did not evaluate higher flows for two reasons. First, the manufacturers recommend against using higher flows because they may affect the performance of the ventilator. Second, higher heliox flow causes higher expense. For these reasons, using higher flows is not practical.

The clinical role of heliox during NPPV is yet to be determined. Although several studies have reported benefit with heliox and NPPV for COPD patients,[13,14] the reported effect was modest. Because these were short-term physiologic studies, the benefit of heliox with NPPV for important outcomes such as avoidance of intubation is not known. It is interesting that these studies were conducted in COPD patients, whereas use of heliox has been more commonly reported for patients with asthma—a disease for which the role of NPPV is unclear. Neither our study nor previously reported studies establish a clear role for heliox with NPPV. However, our results do describe the performance of ventilators for NPPV, which should permit the design of clinical studies to establish the role of heliox with NPPV. Further testing of heliox with NPPV in vivo is needed before a recommendation can be made about the benefit of this therapy.

Conclusions

In conclusion, we found that helium concentrations of about 60% can be delivered using ventilators designed for noninvasive ventilation. With titration of heliox into the ventilator circuit, flows of 18 L/min are required to achieve this helium concentration. Further work is needed to determine the clinical benefit of heliox therapy during noninvasive ventilation.

ACKNOWLEDGEMENTS

We thank Peter Hill PhD for his help in understanding the effects of heliox on NPPV ventilator function. We also thank the manufacturers for providing their ventilators for the study.

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Patient-Ventilator Interactions during Volume-Support Ventilation: Asynchrony and Tidal Volume Instability—A Report of Three Cases

Thierry M Sottiaux MD

During pressure-support ventilation, tidal volume (V_T) can vary according to the level of the patient's respiratory effort and modifications of the thoraco-pulmonary mechanics. To keep V_T as constant as possible, the Siemens Servo 300 ventilator proposes an original modification of pressure-support ventilation, called volume-support ventilation (VSV). VSV is a pressure-limited mode of ventilation that uses V_T as a feedback control: the pressure support level is continuously adjusted to deliver a preset V_T. Thus, the ventilator adapts the inspiratory pressure level, breath by breath, to changes in the patient's inspiratory effort and the mechanical thoraco-pulmonary properties. The clinician sets V_T and respiratory frequency, and the ventilator calculates a preset minute volume. It has been shown that ineffective respiratory efforts can occur during pressure-support ventilation. A mismatch between the neural (ie, patient) and mechanical (ie, ventilator) timings is the main cause of missing breaths occurring while the ventilator is in the inspiratory phase: the reason is that the ventilator does not cycle from inspiration to expiration until the inspiratory flow decreases to a threshold value (5% of the peak inspiratory flow). The patient's ineffective efforts can also occur during the expiratory phase of the ventilator: in that situation, the inspiratory effort occurs before complete lung emptying and is not high enough to trigger the ventilator. The risk of the patient making ineffective efforts is increased by the algorithm included in the VSV mode. If the patient makes numerous ineffective efforts, the frequency of effective efforts (recorded by the ventilator) can be lower than the set frequency, in which case a new target V_T is calculated by the ventilator to achieve the preset minute volume. As V_T increases, the mismatch between the neural and mechanical timings also increases. I report 3 clinical observations showing numerous patient respiratory efforts not sensed by the ventilator and inducing V_T instability during VSV. These ineffective efforts can occur during inspiratory and expiratory phases. The mechanisms are discussed.

Key words: asynchrony, dyssynchrony, patient-ventilator interactions, volume-support ventilation, pressure-support ventilation, tidal volume, inspiratory effort, ventilator triggering, mechanical ventilation. [Respir Care 2001;46(3):255–262]

Introduction

Pressure-support ventilation (PSV) is a patient-triggered, pressure-limited, flow-cycled mode of ventilation. PSV provides a constant level of positive pressure during spontaneous ventilation. Breaths are pressure-triggered or flow-triggered. During PSV, tidal volume (V_T) can vary according to the patient's inspiratory efforts and thoraco-pulmonary mechanics. The Siemens Servo 300 ventilator offers a ventilation mode called volume-support ventilation (VSV). The goal of VSV is to ensure a constant, preset V_T during PSV. Using a closed-loop control system, the Siemens Servo 300 adapts the level of inspiratory pressure support (PS) to deliver a preset V_T. Delivered V_T is used as a feedback control for continuous adjustment of the PS level.[1] The ventilator automatically adapts the PS level to changes in the mechanical thoraco-pulmonary properties and the patient's inspiratory effort. To initiate VSV the clinician sets the target V_T and respiratory frequency.

See The Related Editorial on Page 232

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VSV includes a specific algorithm. On the basis of the preset respiratory frequency and V_T, the ventilator calculates a "minimum minute ventilation" (respiratory frequency multiplied by V_T).[1] If the patient's breathing frequency is lower than the preset frequency, the minimum minute volume cannot be reached. In that situation (minute volume lower than that calculated on the basis of the preset parameters), the ventilator calculates a new target V_T as a reference for regulation of PS. The maximum new calculated V_T may be up to 150% of the preset V_T. For example, with a preset V_T of 500 mL and a preset frequency of 10 breaths/min, the minimum minute volume is 5 L/min. If the patient's frequency drops below 10 breaths/min, the new calculated V_T increases to reach the calculated minimum minute volume and the maximum new calculated V_T will be 750 mL.

During both pressure-triggered and flow-triggered VSV, a bias flow of 2 L/min is delivered into the circuit during expiration. When the ventilator is set to "flow-triggering," the ventilator can be triggered when a flow of 0.7–2 L/min is inhaled by the patient from this bias flow. Flow-triggering is set by moving the trigger button into the green area (lower sensitivity) or red area (higher sensitivity, with the risk of auto-triggering). In this study, we set the trigger button directly between the green area and the red area.

During VSV, the Siemens Servo 300 cycles from inspiration to expiration when the inspiratory flow reaches 5% of the peak inspiratory flow. A safety mechanism limits the inspiratory time (T_I) to a value of 80% of the preset respiratory cycle duration.

Ineffective respiratory efforts may occur during the patient-triggered modes of ventilation, inducing V_T variability.[2] Nava et al[3] and Jubran et al[4] have shown that many patients suffering chronic obstructive pulmonary disease make ineffective efforts during PSV and appear to struggle against the ventilator. The potential for dyssynchronous interaction during VSV has not been evaluated in the literature.

Patient-ventilator asynchrony may escape routine clinical survey. Nevertheless, careful analysis of available respiratory waveforms provided by new ventilators allows the clinician to detect the wasted efforts occurring during the gas delivery phase of the ventilator or during the expiratory phase. Available graphic displays include scalars (waveform plotting pressure or flow or volume vs time) and loops (simultaneous plotting of two respiratory variables).[5,6] Because active use of respiratory muscles may affect the patterns of the curves, patient and ventilator frequencies can be determined by examination of the displayed curves. "1:1 interaction" means that all the patient's breathing efforts trigger the ventilator and induce lung inflations applied by the ventilator. Conversely, "non-1:1 interaction" indicates the presence of active spontaneous inspirations that are not assisted by mechanical inflation.

This report illustrates some examples of patient-ventilator interaction during VSV with the Siemens Servo 300. We observe that patient-ventilator dyssynchrony during VSV can induce marked V_T instability.

Case Reports

Case 1

A woman suffering from chronic obstructive pulmonary disease is admitted to the intensive care unit for hypercapnic respiratory failure. After a short period of controlled mechanical ventilation, we implement gradual weaning with VSV. Just before recording the curves shown in Figure 1, the patient is disconnected from the ventilator for tracheal suction, and then reconnected. After reconnection, return to the preset V_T (0.5 L) is achieved by reactivation of a test breath sequence. The patient's inspiratory effort induces a first test breath (first breath in Fig. 1). The delivered V_T is measured and then the ventilator calculates the inspiratory pressure, providing the desired V_T. This calculated inspiratory pressure is reached using a breath-by-breath pressure increase, in small steps with a maximum change of 3 cm H_2O. The patient's respiratory frequency is 20 breaths/min. Nevertheless, before reaching the calculated level of pressure, the progressive V_T increase finally leads to the occurrence of a missing respiratory effort (arrow 1 on the flow curve). As the V_T increases, the time needed for complete lung emptying increases. Eventually, this results in a level of intrinsic positive end-expiratory pressure ($PEEP_I$) that the patient cannot overcome to trigger the ventilator. The expiratory flow curve almost reaches zero during the patient's inspiratory effort, but without reaching the level of trigger sensitivity. The ventilator cannot recognize the patient's effort and does not apply a positive pressure. Therefore mechanical expiratory time (T_E) is extended and the patient can exhale a larger V_T than previously, evidencing air trapping.

The last breath in Figure 1 is troubling. The patient's effort initiates a breath, and V_T clearly appears higher than during previous breaths. The very large increase in V_T cannot be solely explained by the progressive increase in the PS level applied by the ventilator (see the rate of the breath-by-breath increase in the V_T at the beginning of the sequence). This phenomenon is probably explained by an increase in the "driving pressure for flow." Driving pressure for flow is the difference between the pressure applied by the machine and alveolar pressure. As during the previous expiration, the patient exhales a higher V_T (indirectly evidencing air trapping), end-expiratory alveolar pressure decreases, and consequently the driving pressure increases. Furthermore, this breath is applied for a long time because an end-inspiratory effort (arrow 2) prevents

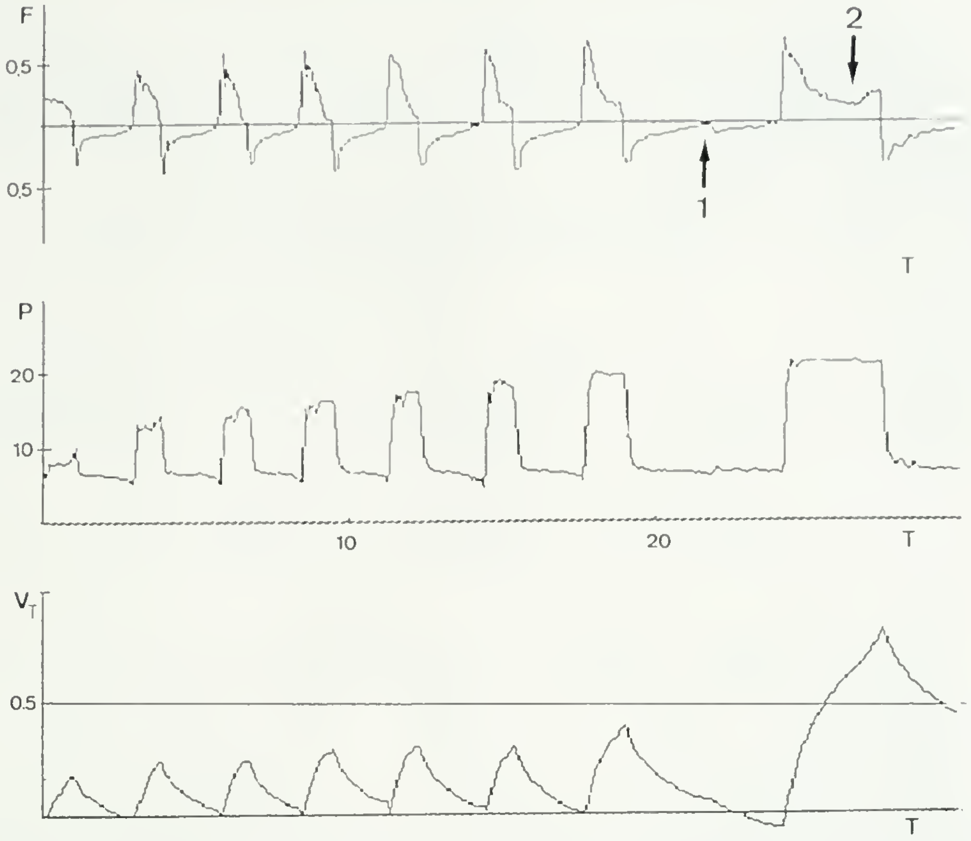

Fig. 1. Curves plotting flow (F, in L/s), pressure (P, in cm H_2O), and tidal volume (V_T, in L) versus time (T, in seconds). The figure shows a test-breath sequence during initiation of volume-support ventilation (VSV). Set parameters are V_T 0.5 L, positive end-expiratory pressure 5 cm H_2O, and frequency 15 breaths/min. The ventilator applies a step-by-step increase in plateau pressure until the target V_T is reached. The patient's breathing frequency averages 20 breaths/min. The information from the inspiratory flow sensor is used to calculate inspiratory V_T. The expiratory flow sensor is used to calculate expiratory V_T. As a consequence, if expiratory V_T is higher than inspiratory V_T, the V_T trace falls below zero. A failed inspiratory effort occurs (arrow 1) because the effort occurs before lung emptying. As a consequence, mechanical expiration time is extended and allows the patient to exhale a higher V_T, evidencing air trapping. During the last breath, a respiratory effort (arrow 2) occurs during the inspiratory phase, preventing the inspiratory flow from reaching the cycling level, so pressurization is maintained and inspiratory V_T is increased well above the target value.

the inspiratory flow from reaching the flow-cycling level (ie, 5% of the peak inspiratory flow). The inspiratory flow curve is upward truncated by the patient's effort. Finally, inspiration is interrupted by the safety mechanism (maximum allowed T_I is 80% of the preset respiratory cycle duration). So, during this breath, the primary flow termination criterion is not met and the ventilator applies the second termination criteria (80% of the cycle time).

This first case illustrates the occurrence of ineffective efforts during a test breath delivered at the initiation of VSV. The test breath per se does not induce the problem. The ventilator progressively increases the applied pressure and, consequently, the V_T. Patient-ventilator interactions can occur during the test sequence if effective T_E is eventually too short to allow a complete exhalation of the inhaled V_T (first arrow in Fig. 1). Relatively high V_T can induce air trapping in the presence of airway obstruction. This case also shows that, during VSV (as well as during PSV with this ventilator), the primary flow criterion (5% of the peak inspiratory flow) induces a relatively long mechanical T_I that can lead to patient-ventilator interaction, as shown in Figure 1 (arrow 2).

Case 2

A patient is admitted after thoracic surgery (left pneumonectomy). The patient's preoperative vital capacity averages 85% of predicted, and his forced expiratory volume in the first second averages 80% of predicted. A pleural tube is inserted perioperatively at the sixth intercostal space. The patient is intubated and ventilates spontaneously. No air leak is detected. VSV is applied with a target frequency of 10 breaths/min and a target V_T of 0.5 L. The patient's respiratory frequency reaches 35 breaths/min while ventilator inflation frequency is 7 breaths/min. Figure 2 shows that the machine/patient frequency ratio is 1/5 (4 out of 5

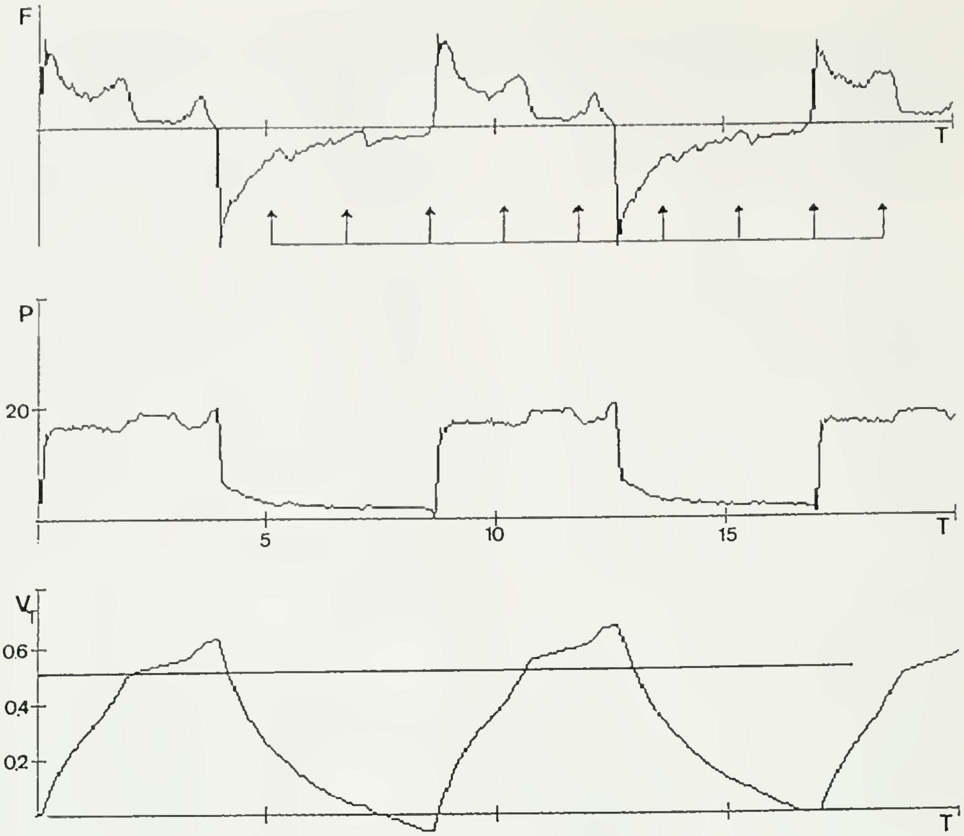

Fig. 2. Curves plotting flow (F, in L/s), pressure (P, in cm H_2O), and tidal volume (V_T, in L) versus time (T, in seconds) recorded after admission of a postoperative (thoracic surgery) patient. Volume-support ventilation (VSV) is applied with a target V_T of 0.5 L and a frequency of 10 breaths/min. The patient's activity is clearly observed on the flow/time and pressure/time curves. The patient's frequency is 35 breaths/min while ventilator frequency reaches 7 breaths/min. The ventilator/patient ratio is 1/5. In this case, the new target V_T averages 0.7 L (see text).

respiratory efforts are missing). Because the ventilator's frequency is lower than the preset frequency, the ventilator calculates a new target V_T based on the preset minute volume. In this case, the new target V_T averages 0.7 L (140% of preset V_T) to guarantee the preset minimum minute volume. We see in this case an illustration of the risk of a vicious circle induced by VSV algorithm: missing breaths are associated with a lower ventilator frequency, and this decreased ventilator frequency induces an increase in the target V_T. This higher V_T induces an increase in the number of missing breaths as a consequence of a higher mechanical timing and an increase in the time needed for complete lung emptying. This patient-ventilator interaction leads to a very low ventilator frequency (T_I = 4.2 s, T_E = 4.25 s) with a high V_T.

This case illustrates the risk of VSV algorithm-induced mismatch between the ventilator and the patient.

Case 3

A patient with no pre-existing pulmonary or chest wall disease is admitted after a stroke. Controlled mechanical ventilation is applied for 24 hours. VSV is initiated with a target V_T of 0.5 L (ie, at the same level as that of the first 24 h of ventilation). Preset respiratory frequency is 15 breaths/min, and PEEP is 7 cm H_2O. Though clinical examination fails to show missing efforts, the flow/time curve analysis evidences a mismatch between the patient's frequency and the ventilator's frequency (Fig. 3). The patient's inspiratory effort frequency reaches 48 breaths/min while the ventilator's frequency averages 12 breaths/min. Therefore, only one out of every 4 respiratory efforts is successful. The analysis of one ventilator cycle (see Fig. 3) shows that:

- The first effort is successful (arrow 1). The expiratory flow trace does not decay to zero at end-exhalation. The flow curve is upward truncated, evidencing air trapping. The patient overcomes the $PEEP_I$ and triggers the ventilator.
- The second effort is ineffective and occurs during the application of the inspiratory plateau pressure, just before the moment of cycling to expiration (arrow 2).
- The third effort (not clearly seen) is failing and occurs at the beginning of expiration (arrow 3).

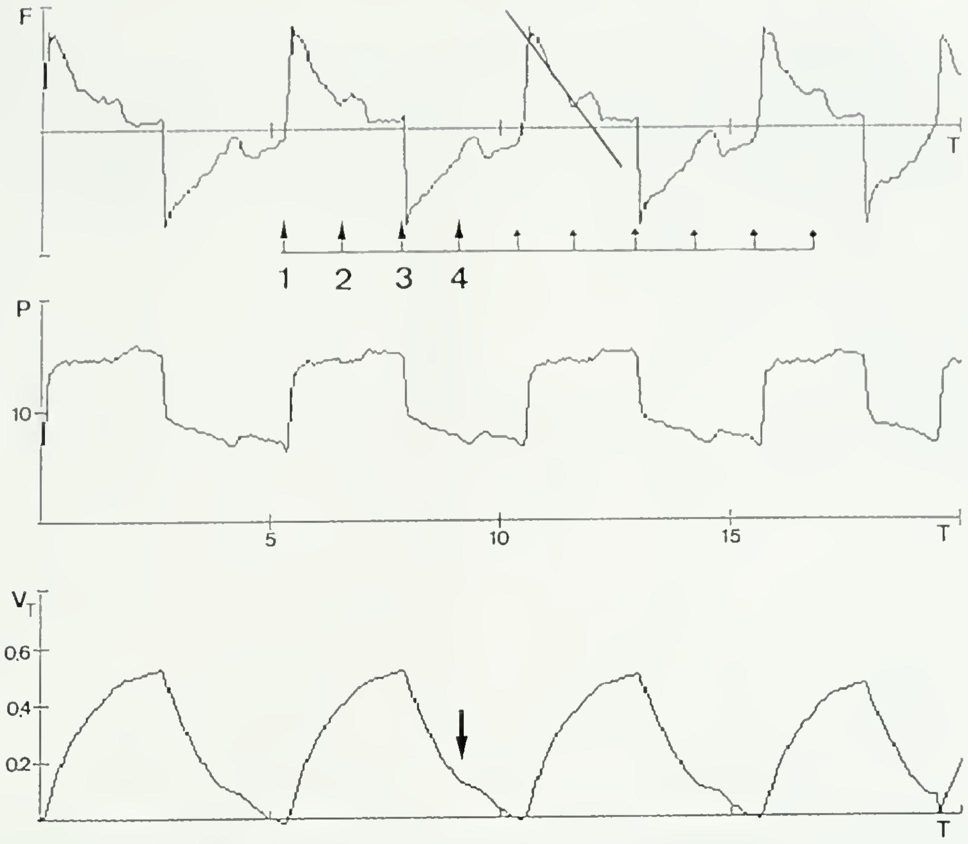

Fig. 3. Flow/time (F, in L/s, T, in s), pressure/time (P, in cm H_2O), and tidal volume/time (V_T, in L) relationships during volume-support ventilation (VSV). The ventilator indicates a frequency of 12 breaths/min. V_T reaches the target value (0.5 L). The flow/time curve clearly shows the true patient's frequency averaging 48 breaths/min. Only 1 out of every 4 breaths triggers the ventilator. Arrow 1: Successful effort. Arrow 2: Effort occurring during mechanical lung inflation and extending inspiratory time. Arrow 3: Effort occurring at the beginning of the expiratory phase. Arrow 4: Effort occurring during expiration, before complete lung emptying (see also the downward arrow on the V_T/T curve).

- The fourth effort is ineffective and occurs during expiration, before complete lung emptying (arrow 4 and downward arrow on the V_T/time curve).

Extrapolating the slope of the decelerating inspiratory flow curve, theoretical T_I would average approximately 1.6 seconds (see extrapolation straight line on the flow/time curve). Nevertheless, as a muscular activity occurs just before inspiratory flow reaches the expiratory cycle criterion (5% of the peak inspiratory flow) (arrow 2), the inspiratory flow curve is upward truncated, delaying the moment of cycling to expiration. True T_I averages 2.5 seconds. This figure shows a situation of patient-ventilator dyssynchrony. The ventilator/patient frequency ratio averages 1/4. Patient respiratory frequency is fixed at a rate of about 48 breaths/min. During the inspiratory phase, before reaching the breath termination flow criterion, the ventilator is insensitive to respiratory efforts. Furthermore, these respiratory efforts, occurring during the inspiratory time, may modify the slope decay of the flow curve and delay the moment of cycling to expiration.

Figure 4 shows the curves displayed one hour later. Target V_T and frequency are 0.5 L and 15 breaths/min, respectively. PEEP is decreased to 4 cm H_2O. The patient is more alert and clinical examination shows respiratory efforts at a frequency of 42 breaths/min. Nevertheless, the ventilator's frequency shows clear irregularities, averaging either 14 (first 2 breaths on Fig. 4) or 21 breaths/min (right side of Fig. 4). Compared with Figure 3, the encroachments on the flow/time curve are more important, evidencing the patient's heavier muscular activity. As a result of the increase in inspiratory muscular activity, the efforts shown by arrow 3 sometimes succeed. This phenomenon is seen on the right part of the figure (see the two circles on the flow/time curve). This sudden efficiency of an inspiratory effort occurring during expiration induces an increase in the ventilator's frequency, with a dramatic decrease of T_E, from 2.4 to 0.75 seconds. So expiration is stopped largely before lung emptying, inducing an increase in air-trapping. During the next breath, because of the increased $PEEP_I$, the same level of applied PS induces a

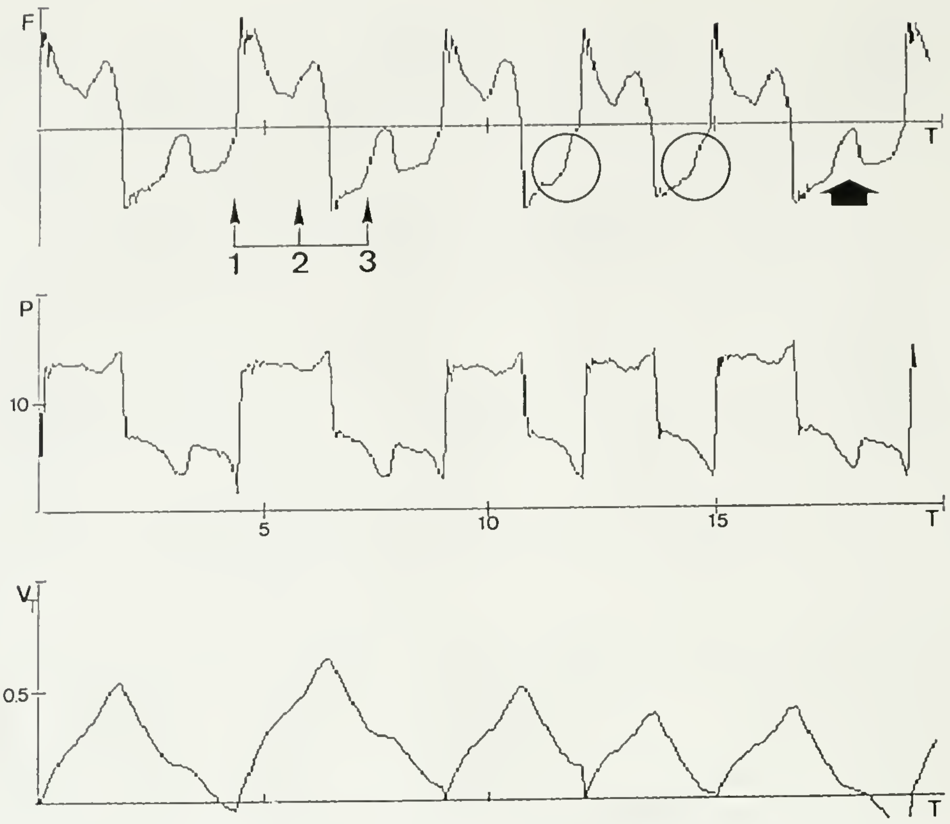

Fig. 4. Flow/time (F, in L/s, T, in s), pressure/time (P, in cm H_2O), and tidal volume/time (V_T, in L) relationships during volume-support ventilation (VSV). These data are from the same patient as in Figure 3, recorded a few hours after the data in Figure 3. Patient clinically showed heavier respiratory efforts. The patient's efforts are shown by the large pressure oscillations. The oscillations on the pressure/time curve indicate that the patient is demanding gas flow in excess of the delivered level of flow. Note that the flow trace never decays to zero at the end of exhalation. The ventilator/patient frequency ratio averages 1/3 in the left side of the figure. In the right side of the figure, the end-expiratory effort becomes successful (see the two circles on the flow/time curve), inducing an increase of the ventilator frequency from 14 (1/3 ratio) to 21 (1/2 ratio) per minute. Finally, this end-expiratory effort becomes ineffective again (large upward arrow).

lower V_T. Finally, because of this air-trapping phenomenon, the breath previously shown by arrow 3 becomes ineffective again (see the large arrow on the flow/time curve in Fig. 4), inducing an increase in the mechanical T_E and allowing greater lung emptying (shown by the decrease in the V_T/time curve below the zero level). In this situation we can observe a high level of V_T change depending on the breath-by-breath air-trapping variability. Interestingly, the heavy respiratory efforts shown by arrow 2 remain totally ineffective and delay the moment of expiratory cycling. Just after this inspiratory effort (arrow 2), the patient begins to forcefully exhale, causing an abrupt decay in the inspiratory flow trace. This expiratory effort can also be seen on the pressure/time curve (increased pressure at the end of inspiration).

This figure illustrates the oscillation of the ventilator/patient frequency ratio (1/3 alternating with 1/2), depending on the intensity of the patient's activity and the moment of occurrence of the neuromuscular stimulus during the expiratory phase. Bignall et al called this phenomenon "chaotic, non-1:1 interaction."[6]

The data in Figure 5 were recorded from the same patient as were the data for Figures 3 and 4. To decrease central respiratory frequency, 10 μg of sufentanil is administered intravenously. Spontaneous frequency decreases to 14 breaths/min and all the patient's inspiratory efforts successfully trigger the ventilator. Spontaneous respiratory frequency is not regular and the increase in T_E between breaths A and B allows the end-expiratory flow to reach zero, with an increase in the exhaled V_T, evidencing air trapping.

Ineffective respiratory efforts shown here are not specifically due to the VSV algorithm and could be observed during either PSV or VSV.

Discussion

To eliminate the problem of V_T instability during PSV, VSV has been proposed. Using V_T as a feedback control

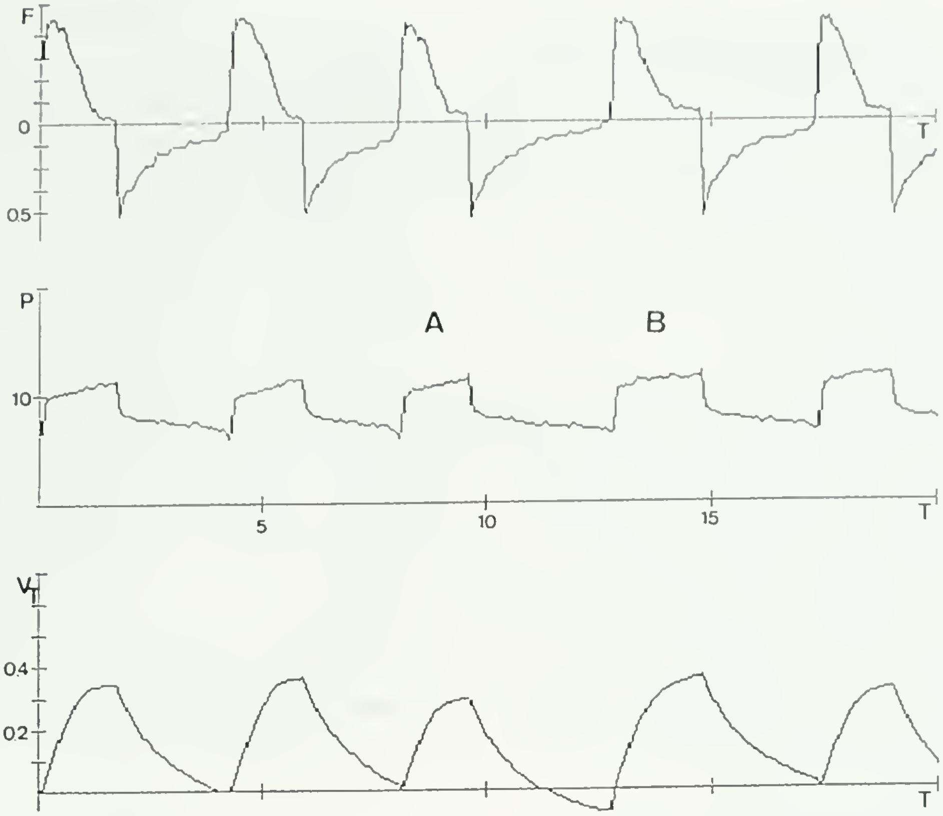

Fig. 5. Flow/time (F, in L/s, T, in s), pressure/time (P, in cm H_2O), and tidal volume/time (V_T, in L) relationships during volume-support ventilation (VSV). These data are from the same patient as in Figure 3, recorded a few hours after the data in Figures 3 and 4. Sufentanil has been administered to reduce respiratory frequency. The patient's breathing frequency averages 14 breaths/min and all the efforts trigger the ventilator. We can nevertheless see that the patient's frequency is not regular. The increase in expiratory time between breaths A and B allows a higher expired V_T.

during VSV, the PS level is continuously adjusted to deliver a constant preset V_T. Nevertheless, clinical experience clearly shows that V_T can be highly unstable in some patients during VSV as well as during PSV. We show here that ineffective efforts occurring during VSV can lead to large breath-by-breath variations of V_T and that this risk could be increased by the VSV algorithm.

Ideal patient-triggered mechanical ventilation is characterised by a 1:1 interaction.[6] Non-1:1 patient-ventilator interaction during ventilatory support indicates the presence of active efforts not sensed by the ventilator. Respiratory efforts may not be sensed by the ventilator when the central respiratory drive activates respiratory muscles during the lung inflation phase or before complete exhalation of the V_T. It has been shown that the respiratory neural stimulus cannot be changed on a breath-by-breath basis.[7] When the neural stimulus frequency is fixed at a relatively high level, the inspiratory and expiratory neural timings are very short, increasing the risk of patient-ventilator dyssynchrony. Ideal coupling between patient effort and machine output is obtained by having neural and mechanical timings equal.[2] Differences between mechanical and neural timings lead to a situation of a patient "fighting" the ventilator, previously called "chaotic mode of interaction."[6] This phenomenon induces an irregular ventilator rate and V_T instability. During PSV (or VSV), the mechanical timing (ie, the duration of the inspiratory phase) is determined by the time taken for the inspiratory flow curve to reach the threshold value for expiration. This mechanical timing depends on the level of the peak inspiratory flow, the flow cycle threshold, and the patient time constant.[2] For example, increases in lung compliance and/or airway resistance (eg, as in a chronic obstructive pulmonary disease patient) reduce the slope of the flow curve decay and, hence, increase mechanical T_I. Before reaching the flow cycle threshold value, the ventilator is insensitive and the patient cannot trigger the ventilator even with a vigorous respiratory effort. As a consequence, mechanical inflation may persist into neural stimulation.[4] Furthermore, because of the patient's inspiratory efforts, the cycling flow threshold may not be reached and the inspiratory time may be prolonged. During PSV and VSV, the Siemens Servo 300 switches from inspiration to expiration when inspiratory flow decays to 5% of the peak inspiratory flow. This threshold value seems too low, promoting an increase in mechanical T_I. During VSV, the risk of patient-ventilator interaction is increased by the algorithm of this particular mode of ventilation. If the rate of the patient's effective efforts falls below the set frequency, the PS level provided by the machine increases to meet the minimum minute volume, so V_T becomes larger than preset. Beyond the risk of induced apnea, this increased V_T can induce or worsen patient-ventilator dyssynchrony. Therefore it may be important to set the machine frequency as close as possible to the patient's respiratory rate.

We also find that many ineffective respiratory efforts can occur during the expiratory phase. T_E may be too short to allow complete lung emptying, resulting in air trapping and inability to overcome $PEEP_I$ to trigger the ventilator. To be effective, inspiratory efforts have to overcome $PEEP_I$ and the preset triggering level.

This observation shows that we have to adapt the level of V_T to the mechanical properties of the system (patient, machine, ventilatory circuit). We have to take into account the time constant of the system. In some situations the lungs will complete inflation or deflation in a relatively long time. High V_T (resulting from high preset inspiratory pressure support) increases the risk of patient-ventilator interaction either during inspiration or expiration.

Is It Possible to Match Neural and Mechanical Timing?

The main goal is to achieve 1:1 interaction, in which case the whole spontaneous activity is used efficiently. Failed efforts may lead to muscular fatigue, discomfort, or agitation. The decrease in the applied PS level has been suggested to decrease ineffective efforts occurring either during inspiration or during expiration. Nevertheless, lowering the PS level leads to a marked increase in respiratory work, with deterioration in blood gas levels.[3] Addition of extrinsic (ventilator set) PEEP has been evaluated.[8] In patient-triggered mode, when ventilatory frequency is governed by the patient's drive, application of extrinsic PEEP (not exceeding $PEEP_I$) may improve patient-ventilator interaction and decrease the rate of missed efforts. Nevertheless, it is important to point out that extrinsic PEEP is not effective when efforts are occurring early in expiration and during inspiration.

Most importantly, the interest of improving the cycling function of the ventilator must be considered. With the Siemens Servo 300, expiration is started when the inspiratory flow decreases to 5% of the peak inspiratory flow. The possibility of adapting this cycle criterion to the spontaneous ventilatory pattern of the patient could improve synchrony between the patient and the machine.

Conclusions

Basically, VSV acts as PSV, except that the amount of applied inspiratory pressure support is calculated by the ventilator to deliver a preset V_T. The specific aim of this new ventilation mode is to avoid the V_T instability usually observed during PSV.

These case reports clearly show that, as during PSV, V_T may not be stable during VSV. This phenomenon can be explained by the occurrence of patient-ventilator interactions during both inspiration and expiration. Patient respiratory efforts that occur during inspiration totally fail because the ventilator does not offer any possibility of triggering during that period. Furthermore, those inspiratory efforts can induce an increase in T_I, with an increase in V_T. Respiratory efforts that occur during expiration can trigger the ventilator only if they are sufficient to overcome the elastic recoil of the lungs. During VSV, the risk of patient-machine interaction is increased by the specific algorithm.

The basic purpose of the Siemens Servo 300 VSV mode is to maintain a steady V_T during a mode of pressure-limited ventilation. Nevertheless, because of the occurrence of patient-ventilator interactions, this primary goal is not always reached.

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Weaning to Extubation Directly from High-Frequency Oscillatory Ventilation in an Infant with Cystic Lung Disease and Persistent Air Leak: A Strategy for Lung Protection

Lori Seller RRT, Krishna Mullahoo RRT, Stephen Liben MD, and Larry C Lands MD PhD

We report the successful weaning and extubation of an infant from a SensorMedics 3100A high-frequency oscillator without returning to conventional ventilation. A 7-week-old term infant with respiratory syncytial virus bronchiolitis complicated by cystic pulmonary lesions repeatedly failed attempts to return to conventional ventilation from high-frequency oscillatory ventilation (HFOV) for weaning, because of recurrent pneumothoraces. A computed tomography of the chest revealed multiple well defined cysts of various sizes involving both lungs. Therefore, weaning to extubation from HFOV was proposed as a way of preventing further air leak. The weaning strategy consisted of a technique we refer to as "sprinting." Using this method, the patient was successfully extubated directly from HFOV, with no complications. A follow-up computed tomography of the chest showed marked improvement in the size of the cystic lesions. The patient was discharged home with no need for home oxygen therapy.

Key words: high-frequency oscillatory ventilation, extubation, sprints, sprinting, cystic pulmonary lesions, ventilator-induced lung injury, weaning. [Respir Care 2001;46(3):263–266]

Introduction

We report the case of a pediatric patient with persistent air leak due to pulmonary cystic lesions and who would not tolerate phasic, conventional mechanical ventilation (CMV). As a result, a weaning strategy was devised to wean from high-frequency oscillatory ventilation (HFOV) to extubation, without returning to CMV.

HFOV is a mode of mechanical ventilation that optimizes gas exchange by delivering tidal volume of less than or equal to anatomical dead space, in combination with supraphysiological frequencies and a constant mean airway pressure.[1]

Pulmonary air leaks are the most commonly reported ventilator-induced lung injury during CMV, occurring at a rate of 5–15%.[2] In the presence of air leak, HFOV has been shown to be an effective and less traumatic method of ventilation, providing a lung-protective strategy.[3,4]

Common clinical practice in weaning from HFOV has been to reinstitute CMV once the acute lung disease has improved. However, neither that nor other standard treatments for pulmonary cysts, including unilateral ventilation and lobectomy, were options for our patient, given the bilateral nature of his lung disease.[5,6]

Our weaning strategy allowed for the progressive evaluation of the patient's ability to breathe spontaneously in conjunction with and eventually without the aid of the oscillator, and allowed us to eventually extubate based on his clinical performance.

Case History

A previously healthy 7-week-old male infant weighing 4.5 kg was diagnosed with severe respiratory syncytial virus bronchiolitis and transferred to the pediatric intensive care unit, intubated with a 3.0 mm uncuffed endotracheal tube. His medical history included a spontaneous

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right-sided pneumothorax at birth, which resolved on its own. On admission to the pediatric intensive care unit, the patient was ventilated on pressure-regulated, volume-controlled mode with a Siemens 300 ventilator (Siemens-Elema AB, Solna, Sweden). Because of a large leak around the endotracheal tube, he was reintubated with a 4.0 mm uncuffed endotracheal tube. On day 6 of treatment he was noted to have decreased air entry on exam. A chest radiograph revealed low lung volumes and right upper and left lower lobe partial collapse. In addition, he required an increase in fraction of inspired oxygen, from 0.40 to 1.0, to maintain S_{pO_2} (arterial oxygen saturation measured via pulse oximetry) > 87%, prompting a switch to HFOV ventilation mode with the SensorMedics 3100A (SensorMedics Corporation, Yorba Linda, California). Initially, using a high lung volume strategy, the patient was placed on HFOV with a frequency of 10 Hz, flow of 15 L/min, mean airway pressure (MAP) of 25 cm H_2O, and an amplitude of 41 cm H_2O. This resulted in a marked decrease in fraction of inspired oxygen need, from 1.0 to 0.60–0.70, while maintaining an $S_{pO_2} > 87\%$.

On day 8 of treatment, a chest radiograph showed large lung volumes as well as a large cyst in the right lower lobe. Because of these findings, a low lung volume strategy (MAP decreased to 20 cm H_2O) and permissive hypercapnia (arterial carbon dioxide tension [P_{aCO_2}] < 60 mm Hg and pH > 7.30) were employed.

On day 9 of admission the patient suffered a large right-sided pneumothorax that required placement of two chest tubes. Because of this acute deterioration, inhaled nitric oxide was started at 20 ppm, with a modest improvement in S_{pO_2}.

From day 9 to day 16, repeated attempts to convert the patient from HFOV to conventional ventilation were complicated by recurrent air leak, always on the right side, despite chest tubes in situ. A computed tomography scan of the chest revealed multiple bilateral pulmonary cysts of various sizes, more evident on the right side than on the left (Fig. 1), which negated possible benefits of unilateral ventilation or selective lobectomy. The patient was therefore maintained on HFOV from day 16 to day 28 of admission in order to minimize pressure swings and the potential for air leak. During those 12 days, his settings were progressively decreased to MAP of 10 cm H_2O, amplitude of 32 cm H_2O, and fraction of inspired oxygen 0.35, and the inhaled nitric oxide was weaned off. On those settings he stabilized and his air leak resolved.

On day 28 of admission, it was decided to attempt to wean the patient from HFOV without a return to CMV, in order to prevent further air leak. On day 31 the chest tubes were removed, with no reaccumulation of air. The first step in beginning the weaning process was to decrease the patient's sedation so that he would start breathing spontaneously above the oscillator. The patient had been on a continuous infusion of sufentanil citrate, which was changed to methadone hydrochloride. The methadone hydrochloride was then gradually weaned. Subsequently, a "sprinting" schedule was devised. "Sprinting" refers to substantially decreasing the ventilator settings for a short period of time, several times a day, to gradually build up respiratory muscle endurance. When not sprinting, the patient returns to baseline parameters to rest. Initial sprinting parameters were MAP of 9 cm H_2O and amplitude of 10 cm H_2O for a period of 20 minutes. By decreasing the amplitude from the baseline of 30 cm H_2O to 10 cm H_2O, we could assess whether the patient's spontaneous respirations would maintain adequate ventilation. Before and after each period of sprinting, the patient's baseline vital signs (respiratory rate, heart rate, S_{pO_2}, and P_{aCO_2}) were recorded. During sprints the patient maintained normocapnia (P_{aCO_2} 35–45 mm Hg). After two days of sprinting, MAP was decreased to 7 cm H_2O for the duration of the sprints, with no change in oxygen requirements. In between sprints, the patient rested on his baseline parameters (MAP of 9 cm H_2O, and amplitude of 30 cm H_2O). It was unclear as to how much the amplitude of 10 cm H_2O was contributing to the maintenance of normocapnia, so eventually the oscillator was stopped for the duration of the sprints, leaving the patient simply on continuous positive airway pressure, yielding no change in post-sprint P_{aCO_2}. As the sprints increased in frequency and duration and the patient began breathing more regularly, it was noticed that he seemed to be working to exhale while sprinting. The 15 L/min bias flow in the HFOV circuit was decreased to 7 L/min during the sprints, to decrease the patient's work of breathing. As the sprinting progressed and the patient continued to do well, the baseline amplitude was weaned gradually, from 32 cm H_2O to 20 cm H_2O. The sprints were gradually increased to one hour by day 37 of admission,

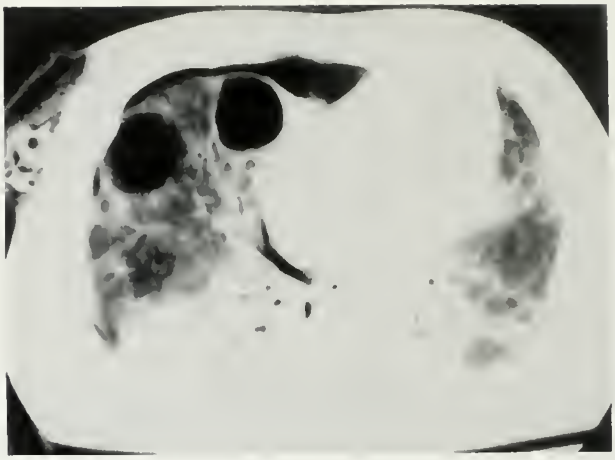

Fig. 1. Computed tomography scan shows diffuse bilateral cysts, more evident on the right side, and right pneumothorax, bilateral air space disease, and air bronchograms.

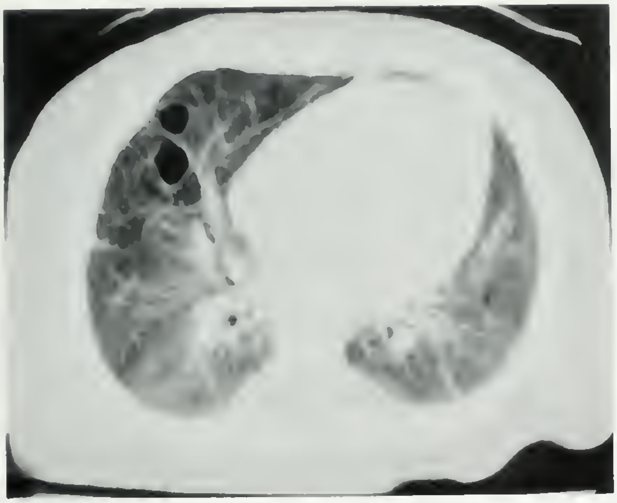

Fig. 2. Follow-up computed tomography scan shows marked decrease in size of cystic lesions.

Table 1 High-Frequency Oscillatory Ventilation Weaning to Extubation Strategy

- Wean sedation to the point where the patient breathes spontaneously but does not actively fight the oscillator
- Perform a pre-sprint evaluation baseline vital signs; air entry with oscillator momentarily stopped; assess for retractions, nasal flaring, or any signs of respiratory distress.
- Amplitude should be decreased to the point where there is no oscillation of the patient's chest (this must be assessed on an individual basis). If the decreased amplitude is well tolerated, the oscillator may be stopped for the duration of the trials.
- Start sprinting for 20 minutes per session, 3 times a day Adjust parameters (flow, MAP, amplitude, and F_{IO_2}) and duration of sprints according to patient tolerance. Allow at least one hour of rest on baseline settings in between sprints.
- As duration of sprints increase (> 30 min), begin weaning baseline amplitude and MAP to the point that oxygenation is stable and lung volumes are maintained on chest radiograph.

MAP = mean airway pressure
F_{IO_2} = fraction of inspired oxygen

leaving the patient breathing spontaneously on a continuous positive airway pressure of 7 cm H_2O. On day 39 of admission, on settings of 10 Hz, 15 L/min, MAP of 9 cm H_2O, amplitude of 20 cm H_2O, the patient was extubated to 0.5 L/min of oxygen via nasal prongs, with no complications. A follow-up computed tomography scan of the chest on day 44 of admission showed marked improvement in the size of the cystic lesions (Fig. 2). The patient was discharged home on day 49, with no need for home oxygen therapy and an almost normal chest radiograph.

Discussion

In order to avoid ventilator-induced lung injury, new modes of ventilation and new clinical practice guidelines have emerged.[2,7–9] Early experimental studies of HFOV used for diffuse alveolar disease showed less acute barotrauma and more uniform lung inflation.[10] Further studies showed HFOV to be effective in the treatment of air leaks, because of the smaller alveolar pressure swings.[3] Although HFOV is not typically used in diseases affecting the small airways, one study has reported beneficial use of HFOV in bronchiolitis.[11] Ideally, HFOV should be started early in the course of hypoxic respiratory failure and/or air leak, as a lung-protective strategy.[12]

Cystic pulmonary lesions during mechanical ventilation present a ventilatory challenge in that they increase the risk of air leak, compress surrounding healthy alveoli (leading to increased dead space ventilation), and can lead to pooling of secretions. Whether pulmonary cysts occur secondary to mechanical ventilation or infectious processes or are congenital in nature, standard treatments include unilateral ventilation and possible lobectomy.[6]

Our weaning strategy allowed for the progressive evaluation of the patient's spontaneous ventilatory capability, while avoiding the higher peak airway pressures and possible patient-ventilator dyssynchrony associated with CMV. Effective tidal breathing adequate to maintain normocapnia can be achieved by a pediatric patient on HFOV by decreasing the MAP and the amplitude to the point of minimal or no oscillation. Although there are a few reports of successful weaning directly from HFOV, only one contained any details as to how this was accomplished.[7,11,13,14] Therefore we developed our own weaning schedule (Table 1).

We speculate that HFOV may have been a successful strategy in this case, because the smaller alveolar pressure swings allowed the recurrent air leak to resolve. Weaning from HFOV may therefore be a feasible option for patients who require a lung-protective strategy throughout the course of mechanical ventilation.

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Innovations in Mechanical Ventilation: What Are (and What Should Be) the Drivers?

Neil R MacIntyre MD FAARC

Introduction

Before I begin, I would like to make a few comments about the man for whom this lectureship is named. It is a real honor for me to be in this position, giving the Roger Bone Lecture this year. I knew Roger for a number of years. He was a superb clinician, an outstanding investigator, and a true leader in our profession. He was a Master Fellow of the College [American College of Chest Physicians] and one of the most prolific scientific writers of our time. Indeed, several of my own publications were ones on which I had the honor to share authorship with Roger. So it's very special for me to be here with Roger's family and my own family to recognize his accomplishments and his memory.

The topic I chose for this morning is the process of how innovations are introduced into clinical practice. Although I'm focusing on innovations in mechanical ventilation, the underlying concepts are applicable to the introduction of any innovation—be it a revolutionary new device or simply a new way to use an established technique. So what I thought I'd do with you this morning is to give you my perspective as to how things move from a germ of an idea to actual clinical practice. Specifically, I want to address the question, "What are the drivers for innovation implementation, and what forces are involved in taking an idea to fruition in our clinical practice?"

Before I go further, I think it is useful to remind ourselves that the basic concepts underlying mechanical ventilation are not new. Indeed, centuries ago simple bellows systems were used to resuscitate drowning victims or workers overcome by fumes or smoke. Since that time, a number of innovations have helped evolve this basic concept into what we now call modern mechanical ventilatory support.[1] Oxygen was first identified by Joseph Priestley several hundred years ago. However, I think the real beginnings of modern mechanical ventilation began with the iron lung at the beginning of the 20th century, during the polio epidemics. Following this, in the 1930s and then into World War II, the development of artificial airways and positive pressure anesthesia ventilators were major steps forward in providing life support to patients in respiratory failure. The addition of positive end-expiratory pressure, first described by Barach in the 1930s, became widespread in the 1960s following Petty and Ashbaugh's description of the acute respiratory distress syndrome and the ability of positive end-expiratory pressure in this syndrome to improve gas exchange.[2] In the 1970s and into the 1980s, microprocessors became much more widely available to govern gas flow and to allow these devices to respond to patient demand. This permitted patients and ventilators to interact, thus reducing the need to heavily sedate or paralyze patients to provide patient-ventilator synchrony.

Over the last decade, there has been an explosive awareness of ventilator-induced lung injury.[3] This injury occurs both when lung units are distended beyond their physiologic maximums as well as when collapsed lung units are subjected to repeated opening and closing shear stresses. Importantly, these injuries not only produce physical lung damage but also release inflammatory cytokines into the systemic circulation and promote translocation of bacteria from airways/alveoli into the systemic circulation. The importance of this injury was driven home by the results of the National Institutes of Health Acute Respiratory Distress Syndrome Network ventilator management trial, which showed a dramatic 25% reduction in mortality in patients managed with a low tidal volume (V_T) strategy designed to limit overdistention.[4]

The use of mechanical ventilation is growing in an almost linear fashion. Data from my own institution at Duke University have shown an approximate doubling of ventilator hours every 8 years since I have been there. There are many forces driving this. First, the population is getting steadily older. Older populations require more medi-

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cal care. Second, more aggressive medical therapies are being offered. Oncology is a classic example: more aggressive oncologic regimens are being used, patients are becoming sicker with septic episodes, and we're able to get them through that. Third, in the surgical arena, the same thing is happening: more aggressive surgeries, coronary bypass grafting in patients in their eighties and nineties, transplantation—all of these things are driving up the need for mechanical ventilatory support. I fully expect it to continue to rise in a linear fashion for the foreseeable future. The good news is there's going to be a job for us!

Innovations Are Needed

Despite the overwhelming success of mechanical ventilation in providing life support to millions of patients, innovations are clearly needed. This technology is by no means perfect. Table 1 is a list of some of the important problems that I think exist right now with our mechanical ventilators. I've already touched on the issue of ventilator-induced lung injury: inappropriate overstretch and inappropriate under-recruitment. In addition, patients are still often uncomfortable when we're trying to use interactive modes. We are forced to use things like sedatives in unacceptably high amounts to keep patients from "fighting" the ventilator, and this often serves only to delay their removal from the ventilator. Infections are a big problem. The longer a patient is on a ventilator, the greater the risk of ventilator-associated pneumonia. I think we need better ways of assessing patients in terms of how fast we can withdraw them from ventilatory support. We need to be aggressively removing the ventilator when it's no longer needed. Another important issue is artificial airway integrity—keeping the patient safely attached to the ventilator. That may sound like a trivial item, but undetected patient ventilator disconnects can cost millions of dollars in lawsuits. We also need to minimize adverse cardiovascular effects of positive pressure ventilation, which has been known for years to compromise cardiac filling; there are ways of actually using the positive pressure breath to sometimes *help* the cardiovascular system. And finally, there is always the potential for human error in providing inappropriate breath delivery settings and inappropriate monitoring/alarm parameters.

Table 1. Aspects of Mechanical Ventilation in Need of Innovations

- Ventilator-induced lung injury
- Comfort/imposed loads and consequent sedation needs
- Infections
- Assessments to guide timely increased/decreased support
- Airway integrity
- Hemodynamic compromise
- Human error on settings/alarms

This is obviously not a complete list, but it's a list of the things that I think are the most important. I always like to keep that list in mind in addressing a new innovation put forth by a clinician, manufacturer, or engineer. The first question should always be, "Which of these problems is the innovation addressing?" Innovation development *should* be driven by these clinical problems.

Forces (Drivers) Influencing the Development of Innovations

Although problem-solving the issues of Table 1 should be the major driver of innovations, there are a number of other forces at work. First, there is the creativity of innovators, both clinicians and engineers. Second, there are the marketing efforts of manufacturers. Third, there are regulatory concerns. And fourth, there are payer issues regarding costs. Let me go into each of these in just a bit more detail.

The creativity of innovators, both clinicians and the engineers, is an important driving force. Many creative individuals want to develop ideas simply because they can be done. In the field of mechanical ventilation, new ways to manipulate gas delivery patterns, feedback controls, and new parameters to monitor are all examples of potential innovations that may or may not have relevance to the problems of Table 1. Nevertheless, they can be exciting intellectual challenges that reward the innovator with a certain satisfaction just for having done it. Unfortunately, once the innovation is developed, the excitement of the innovator for his/her concept (along with accompanying peer recognition and potential monetary rewards) can drive implementation even in the absence of obvious clinical relevance or supporting data.

Ventilator manufacturers work within a market of several hundred million dollars per year for new equipment.[5] To increase market share, their products must be viewed as having more value than those of their competitors. Although most manufacturers would like to provide real value, increased sales are often driven by perceived value. Although perceived value can have some relationship to real value, "expert" testimonials, overinterpretation of limited data, and strong emphasis on "concept selling" can create perceived value and drive the implementation of innovations that have not been adequately established as providing real value.

The regulatory agencies are charged with assuring safety and efficacy of new devices. In the United States, the Food and Drug Administration has responsibility for assuring safety and efficacy of medical devices. This is done through two processes. One is the "510(k)" process (so named because it refers to a specific section in the law) and merely requires the manufacturer to show "substantial equivalence" to a device already on the market; the other resembles a new drug application and is referred to as a pre-market application. Requirements to meet regulatory standards can serve to slow down innovation development. These requirements, however, can also spur innovative efforts to improve safety, enhance documentation, and limit liability.

Finally, payer concerns are focused on costs—both capital and operational. On one hand, these concerns can be an important driver to develop more cost-effective equipment. On the other hand, limited research and development resources can be a substantial deterrent to innovation.

Assessing Claims Made about an Innovation

Claims about an innovation drive the decision to implement the innovation. Claims can come from any of the 4 perspectives listed above (inventor, manufacturer, regulator, or payer), but it is ultimately the clinician/caregiver who should be responsible for deciding the value of an innovation. Proper assessment of claims is thus critical. Unfortunately, limited data, superficial analyses, misunderstanding and/or inappropriate extrapolation of study results, and the limitations of the regulatory process can make this task very difficult. Some specific examples follow.

Inappropriate Extrapolation of Design Concepts and Engineering Results into a Clinical Claim

A good example of this involves new mechanical ventilation modes that provide automatic feedback of ventilator parameters[6] (eg, pressure-regulated volume control, volume support, and adaptive support ventilation). While each of these modes has been shown in engineering testing to perform as designed, no outcome data showing benefit exist. Indeed, there may even be situations where outcome may be adversely impacted (eg, if the target V_T with volume support is set too high, weaning may be inappropriately delayed). Despite this, claims are frequently made by enthusiastic users that these modes can automatically wean patients—claims derived only from the design concept (however flawed) and engineering testing.

Misunderstanding of a Design Concept

I would like to offer two examples of this. First, claims are made that pressure-regulated volume control can provide *both* pressure-limited and volume-guaranteed breaths. Unfortunately, that is impossible: the laws of physics and the equation of motion do not allow it. If compliance changes, if resistance changes, if mechanics change, you cannot keep pressure *and* volume constant. Something is going to have to give, and indeed pressure-regulated volume control will add pressure as necessary or sacrifice volume as necessary within clinically determined ranges. A second example is airway pressure release ventilation (APRV). The claim is made that APRV reduces maximum lung pressures and thus provides lung protection. Although lower maximum pressures probably do reduce overdistention injury, APRV does not necessarily reduce maximum airway pressures. Indeed, if no spontaneous breaths are present and comparable levels of ventilatory support and positive end-expiratory pressure (either applied or intrinsic) are provided, APRV has identical maximum airway pressures to standard pressure control ventilation—the reduced maximum airway pressures are thus seen *only* when spontaneous breathing is present and APRV is compared to synchronized intermittent mandatory ventilation.

Inappropriate Extrapolation of Intermediate End Points

Intermediate end points refer to measurements (usually made at the bedside) that indicate a physiologic response to an innovation under investigation. Examples would include arterial blood gas tensions, airway pressures, and breathing frequency. While certainly useful markers of effect, they are not necessarily indicative of a beneficial clinical outcome such as survival, ventilator duration, length of stay, or complication rates. Indeed, improvement in intermediate end points may sometimes be associated with *worse* clinical outcomes. I would like to illustrate 3 im-

Table 2. Examples of End Points for Different Levels of Efficacy Assessment

Level I—Engineering Assessment
- Valve performance (sensitivity, responsiveness)
- Monitor accuracy
- Flow capabilities
- Ease of operation
- Noise
- Durability
- Cost of operation

Level II—Physiologic Assessment
- Ventilation (total, alveolar, dead space)
- Gas exchange
- Cardiac function (filling, contractility)
- Respiratory system mechanics
- Muscle function (drive, work, pressure-time products)
- Air trapping (intrinsic positive end-expiratory pressure)

Level III—Clinical Outcome Assessment
- Overall survival, long-term survival
- Ventilator days, intensive care unit days, hospital days
- Days alive off ventilator
- Incidence of important complications (barotrauma, pneumonia)

(From Reference 9.)

Table 3. Proposed Efficacy Requirements

Incremental Risk Increase (Regulatory/Clinician Issues)	Incremental Cost Increase (Payer Issues)		
	Minor	Moderate	Major
Minor	Level I	Level II	Level III
Moderate	Level II	Level II	Level III
Major	Level III	Level III	Level III

Level I = Engineering end points
Level II = Physiologic (intermediate) end points
Level III = Clinical outcome end points
(From Reference 9.)

portant examples of this. First, a decade ago, it became apparent that targeting a normal arterial carbon dioxide tension in an asthmatic often produced very high airway pressures and lung injury.[7] "Permissive" hypercapnia was the follow-up notion that allowing an intermediate end point (arterial carbon dioxide tension) to worsen resulted in better outcome.

Second, in the National Institutes of Health Acute Respiratory Distress Syndrome Network ventilator management trial,[4] the high-V_T group (which had the highest mortality) actually had the *best* P_{O_2}/F_{IO_2} ratio (ratio of partial pressure of oxygen to fraction of inspired oxygen) over the first 2 days of the study.

Third, in a large group of medical intensive care unit patients, increasing cardiac output with dobutamine clearly improved oxygen delivery, but it was associated with a higher mortality.[8] The important message is to beware putting excessive importance on intermediate end points as surrogate markers for clinical outcomes.

Unrealistic Expectations of the Regulatory Process

The regulatory process has two important limitations. First, with a 510(k) application, an innovator submits engineering (and sometimes clinical) data that support a claim of "substantial equivalence" to previous devices. If the Food and Drug Administration accepts that claim, innovators are free to market the innovation. Interestingly, although the manufacturer of the innovation can only officially claim substantial equivalence, it is often not long before a number of enthusiastic supporters begin touting the innovation as something quite different from previous devices. Of note is that since 1976 (the initiation of Food and Drug Administration responsibility over devices), every innovation in mechanical ventilation, outside of the high-frequency ventilator, has been approved through this process. Pressure-regulated volume control, adaptive support ventilation, pressure support, and so forth have all only gone through this so-called "substantially equivalent" process. The second limitation is that regulatory agencies have minimal authority to regulate innovative *uses* of currently approved devices. That is to say, you can take that ventilator, even though it was studied only under certain modes and conditions and V_T ranges, and do whatever you want with it: lengthen inspiratory time, shorten inspiratory time, increase pressures, lower pressures—there are no regulatory issues on how you use an approved device. In summary, then, the regulatory process provides some help in establishing the value of an innovation but certainly cannot be the only source of information for a clinician.

What Is Needed to Establish the Value of an Innovation?

So what should we ask of innovators before we take their devices, their gadgets, their new strategies and start to incorporate them into our practice? This question has been explored in a number of consensus conferences,[9] and some important concepts have evolved from these that I think are helpful in making sense of this process.

In general, the claim of benefit should be commensurate with the cost and the risk of the device or the innovation. Accordingly, we ought to expect different levels of efficacy testing based on these factors. Fundamentally, there are 3 different types of claims and levels of efficacy testing. Level I claims concern engineering or user convenience benefits. Level II claims concern physiologic (intermediate end point) benefits. Level III claims concern important clinical outcome benefits. Examples of the 3 levels of efficacy testing are given in Table 2, and the linkage of claim/required efficacy testing to risk/cost is illustrated in Table 3. On the horizontal axis in Table 3 are incremental cost increases; on the vertical axis are incremental risk increases. Payers are obviously interested in

Table 4. Estimated Risk/Cost Impact of Recent Innovations

Innovation	Consensus Opinion	
	Risk	Cost
Pressure-slope adjustment	Minor	Low
Inverse-ratio ventilation	Major	Low
Tracheal gas insufflation	Moderate	Moderate
Proportional-assist ventilation	Moderate	Moderate
Extracorporeal respiratory support	Major	High
Flow-triggering	Minor	Low
High-frequency ventilation	Major	High
Esophageal pressure monitoring	Minor	Moderate

(From Reference 10.)

Table 5. Levels of Efficacy Data Available

Innovation	RC Ed Board Recommends	Levels of Available Efficacy Data I	II	III
Flow triggering	I	+	+	
Pressure slope	I	+	+	
Proportional assist	II	+	+	–
Tracheal gas insufflation	II	+	+	
Pressure-regulated volume control	II	+	–	
Volume support	II	+	–	
Pressure controlled inverse ratio ventilation	III	+	–	
High-frequency ventilation (neonates)	III	+	+	+
Extracorporeal membrane oxygenation (adults)	III	+	+	

RC Ed Board = Editorial Board of RESPIRATORY CARE Journal
Level I = Engineering end points
Level II = Physiologic (intermediate) end points
Level III = Clinical outcome end points

the former; clinicians and regulators are interested in the latter. Using this approach, innovations with low risk and low cost could be considered beneficial even if only engineering improvements or user convenience was claimed (and verified with engineering testing). In a similar fashion, if costs or risks are moderate or less, an innovation could be considered beneficial even if only an intermediate end point benefit is claimed (and verified with clinical testing). However, if a major risk or cost is involved, a claim has to be made that the innovation improves an important clinical outcome, which then has to be proven in a clinical outcome study.

In 1995 I was on the Editorial Board of RESPIRATORY CARE Journal and I surveyed the other members of the Board for their assessment of risk and cost of a number of mechanical ventilation innovations over the previous 10 years. The consensus opinion was remarkably consistent and is summarized in Table 4.[10] With the data, I then established what level of claim and testing should have been done using the Table 3 approach. The results are summarized in Table 5. As can be seen, innovation acceptance historically has not always followed the recommendations of this grid. Certainly some high-risk/high-cost items such as high-frequency ventilation have been evaluated properly, but a number of other innovations have not. I think this needs to be a wake-up call to all of us to demand proper assessments of innovations before they enter clinical practice.

Conclusions

Let me conclude with a few broad statements. First, innovations are needed. There is a long list of things that are not right with ventilators yet. Clinical problems such as overstretch injury, nosocomial infection, costs, and inadequate monitoring/alarms clearly demonstrate the need for innovations.

Second, there are many forces (drivers) at work to produce innovations. In addition to solving clinical problems, the creativity of the innovator, the marketing strategies of manufacturers, the safety/liability issues of regulators, and the cost concerns of payers all influence the development of innovations.

Third, innovators need funding sources to encourage both design development *and* proper assessment testing.

Fourth, before an innovation is accepted as beneficial to clinicians, the efficacy claim and required proof should be established commensurate with the risk and cost of the device.

And then, finally, my more sobering conclusion is that, unfortunately, while some innovations have been properly assessed, many others have not.

So with that I will conclude. I again want to thank the College for this honor to be up here in the name of Roger Bone, a very dear friend both of myself and of the respiratory profession. Thank you all for your attention.

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Letters

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Device Evaluation: A Critique

A good portion of the research published in Respiratory Care involves device evaluation studies. Therefore, it is important that the appropriate study designs are used and that the hypotheses and conclusions are appropriately matched to those designs. A recent article in this journal highlights some of the problems that can arise.

Raake et al[1] evaluated a blood gas monitor in terms of its ". . . accuracy and precision. . ." and concluded that the system provided ". . . accurate blood gas results. . . ." Studies of accuracy, by definition, require that measured values be compared to some type of "gold standard" values.[1–5] The gold standard for blood gas measurement is whole blood tonometry, using blood prepared to specific values with precision gas mixtures. In contrast, Raake et al compared measured values from the bedside monitor with values from duplicate samples assayed with the standard laboratory analyzer. Then they used the data analysis method described by Bland and Altman[6] (mentioned in the illustrations but not listed in the references) to display the "bias and precision" along with some regression plots.

The main problem with the Raake et al study is that it employed a design appropriate for the evaluation of *agreement between two methods*, not for determining the *accuracy of a new device*. Bland and Altman never even mentioned the terms accuracy and precision in their report. The idea behind evaluating agreement is to estimate the systematic error by calculating the mean difference between the new device and the conventional device. Then the random error is expressed as the terms of the standard deviation of the differences. The resultant total error or "limits of agreement" are then calculated as the mean difference ± 2.0 standard deviations.

It is not entirely clear that the Raake et al data were analyzed using the Bland and Altman methods. Raake et al state that "bias was defined as the mean difference between Sensicath System and Corning blood gas values," which is correct, but that "precision was defined as the standard deviation of the *mean* difference between. . . measurements" (emphasis mine). The standard deviation of the mean is the standard error and differs from the standard deviation of individual values by a factor equal to one over the square root of the number of data points (ie, standard error is much smaller than standard deviation). They also used regression to compare the measurements, but Bland and Altman state that "in the analysis of measurement method comparison data neither the correlation coefficient nor techniques such as regression analysis are appropriate," and they explain why in great detail, with examples.[6]

If we can overlook the inconsistency of hypothesis, conclusion, and design, and assume that the data were indeed calculated correctly, and ignore the irrelevant data analyses, is there any way we can accept the conclusion stated in Raake et al's abstract: "When compared to a Corning 855 blood gas analyzer, the Sensicath System was found to provide acceptable blood gas values. . . "?

First of all, as explained in detail elsewhere,[2–5] acceptable differences between new and conventional measurements must be established a priori. These standards can be derived a number of ways, but essentially one must decide if the expected difference will be of clinical importance (ie, that the clinical decision might change depending on which instrument was used for the measurement). For example, in our blood gas laboratory, the maximum acceptable difference in partial pressure of oxygen (P_{O_2}) values on a split sample measured on two similar machines is 7 mm Hg. Thus, it would be reasonable for our lab to demand that any new device provide individual measurements that are within 7 mm Hg of measurements from the lab analyzers, if our standard of care is not to be affected. This type of reasoning has been used in other studies of similar devices.[7] Once the standard is set, a statistical analysis is required to evaluate the probability that future measurements on the new device will be within that range.

The "limits of agreement" analysis describes the expected difference in individual measurements, not the expected mean difference. That is because we make clinical decisions at the bedside based on individual measurements. We do not, for example, take several blood gas samples, calculate the average P_{O_2}, then make a ventilator change. Thus, analyzing comparison data in terms of confidence intervals for the mean difference (bias) is not relevant. It is even less appropriate to make any conclusion about a point estimate of bias (ie, a single value of the mean difference). Yet Raake et al do just that when they state that "The P_{O_2} measurement bias was slightly greater (−4.5) but still clinically unimportant." If we are going to use the limits of agreement method, then our conclusions must be based on what that method shows. For P_{O_2}, the data in the Raake et al report show limits of agreement ranging from −28.1 mm Hg to 19.1 mm Hg. This means that an individual measurement of P_{O_2} on the Sensicath system could be as much as 28 mm Hg different from an identical sample analyzed on the Corning machine! Surely that is clinically relevant. Even on P_{O_2} values less than 50 mm Hg, the expected difference could be as much as 12.8 mm Hg. Similarly, the pH could be as different as 0.09 and the carbon dioxide tension could differ by 12.8 mm Hg. In fact, the expected error intervals are even greater than this. The limits of agreement were estimated as ± 2.0 standard deviations, whereas, accounting for the relatively small sample size, it should have been ± 2.4 standard deviations.[2–5] (Note that there is a typographical error in Table 3 of the Raake et al report, where P_{CO_2} is labeled P_{O_2}).

If our laboratory found this much difference between two supposedly similar machines on a single split sample (for any of the 3 analytes), the machines would be immediately removed from service and fixed. Therefore, I disagree with Raake et al about their conclusion of the acceptability of the Sensicath system as a substitute for a laboratory analyzer.

Raake et al point out that there are no official standards for agreement between old and new measurement devices. Thus, they suggest the use of the CLIA (Clinical Laboratory Improvement Amendments of 1988) criteria for acceptable performance.[8] However, those criteria were meant for comparing a laboratory's performance with 10 or more refereed laboratories. There are more sources of error between laboratories than within a single laboratory, so those criteria would be too loose for assessing the acceptability of a new device introduced to the practice of one hospital. CLIA standards, under section 493.1213(b), state that "Although no specific guidelines exist for verifying a test method, each laboratory is responsible for determining the performance characteristics of its own methods. . . " and ". . . verification may be accomplished by comparison of split sample results with results obtained from a method that has been shown to provide clinically valid results." Since the College of American Pathologists guidelines for blood gas laboratories recommend split sample testing among similar analyzers as an ongoing quality control measure, laboratories approved by the College of American Pathologists should already have the type of agreement data I mentioned above. These are the criteria each lab should use in determining whether to accept new technology.

It is easy to be seduced by the convenience and speed of bedside measurement devices, especially when they offer other advantages such as reduced blood loss. But if we sacrifice scientifically based decisions for such assumed benefits, we may pay hidden costs in the long run. For example, suppose we did accept less precise blood gas measurements. Might that render ventilator and drug therapy decisions less precise and hence increase length of intensive care unit stay and hospital cost, not to mention increase adverse reactions? It seems to me that it would be a lot more practical to avoid this possibility by maintaining accuracy standards now than to try to prove in the future that reducing standards does no harm.

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Allen's Test: Fact or Myth?

Whether the Allen's test should be performed prior to obtaining an arterial blood gas sample from the radial site continues to be an area of controversy. Although many institutions have policies indicating the need to perform the Allen's test, these are not adhered to or are frequently ignored. Until recently, our institution's policy required the Allen's test when the radial artery was chosen for blood gas sampling. When a positive Allen's test (that is, failure to demonstrate adequate filling within six seconds) was observed by the respiratory therapist, it would be documented in the medical record. Because of the controversial nature of this policy, it was decided by the medical directors of the clinical laboratory and pulmonary medicine and by respiratory care management that both a literature review and a study would be undertaken.

SEE THE RELATED EDITORIAL ON PAGE 229

When searching the literature there were two articles that led us to believe that the Allen's test was not a valid indicator or predictor of hand ischemia. Wilkins[1] describes that the Allen's test does not appear to be a valid predictor of ischemia injury to the hand following radial artery cannulation. It might therefore be argued that it is wrong to deny patients the benefits that may accrue from radial artery cannulation, in spite of a positive Allen's test if these tests are performed. A study performed by Slogoff et al[2] concluded that, in the absence of peripheral vascular disease, the Allen's test is not a predictor of hand ischemia during or after radial artery cannulation, that when decreased or absent radial artery flow follows cannulation it is of no clinical consequence, and that radial artery cannulation is a low-risk, high-benefit monitoring technique that deserves wide clinical use.

Our study, which was performed in 1999, randomly reviewed 300 arterial blood gas samplings in which the Allen's test was performed. The results revealed that only two patients, or 0.7%, presented with a positive Allen's test, indicating decreased collateral circulation to the hand, of which one was obviously due to surgical repair of the wrist, which had a direct effect on the ulnar artery circulation.

In conclusion, it appears that a positive Allen's test is both rare and may not be a valid predictor of a negative outcome of radial artery puncture. Therefore, it was decided that the Allen's test would no longer be routinely performed at our institution unless the patient had had an injury or surgical procedure to the site of the wrist. Even in that circumstance the test may be of limited clinical benefit, based on the above. Certainly it would be of tremendous importance if further studies were performed so that clinicians could make rational decisions about whether this procedure is necessary before obtaining a radial artery blood sample.

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Aerosols and the Profession of Respiratory Care: Leading the Way Out of the Fog

In the June[1] and July[2] 2000 issues of RESPIRATORY CARE are presented the most detailed and up-to-date information ever published on aerosols and delivery devices. The work of the Consensus Conference and New Horizons Symposium members and authors is to be commended, and the respiratory care profession should be proud of its part in such a momentous work.

One area of importance within the administration of aerosols is the selection of the delivery device. In the Consensus Statement, a fascinating statement is made: "pMDI [pressurized metered-dose inhaler] and DPI [dry powder inhaler] systems are underutilized in the United States in the acute care setting."[3] One barrier deserves particular consideration because it concerns the profession of respiratory care. That barrier is the misconception on the part of clinicians concerning the efficacy of those devices in the acute care setting. It is my observation that we, as a profession, may have contributed to the underutilization of the pMDI and DPI systems.

First, what is the relationship between the respiratory care profession and aerosol delivery in the United States? The United States is the birthplace of the respiratory care profession. It has a primary role in the administration of aerosols and the selection of delivery devices in the acute care setting. One could say this observation is too simplistic and lacks data to support its impact on the problem. However, it is true that the United States is both unique in the problem identified by the committee and in its relationship with the respiratory care profession. One could argue that all other nations in the world have not benefited from the expertise of the profession, and therefore they overutilize certain devices. However, such an argument would ignore logic and statistical probability, and it would be arrogant. The international experts of the Consensus Conference do consider it an important observation; therefore, we as a profession must address why the United States lags behind the rest of the world in this area.

A second observation closely related to the first concerns a common problem whenever an attempt is made to convert from nebulizers to the pMDI system in the acute care setting. In many institutions there is little or no difference in the cost of the delivery methods.[4] This is supposedly because of the labor required to do a pMDI treatment; however, most if not all clinicians will readily admit that a pMDI can be administered in substantially less time that a nebulizer, and there is even data to support this clinical reality.[5,6] Unfortunately, this type of discrepancy in labor requirements may have been perpetuated by the profession's own labor calculating system: the Uniform Reporting Manual.[7] The Uniform Reporting Manual suggests that there is almost no difference in labor requirements between the delivery devices (12 minutes vs 11 minutes). This manual is highly respected and enjoys widespread use in establishing labor requirements. The labor requirements are then translated into the cost of various procedures performed by respiratory therapists (RTs). The labor value assigned, and thus the cost, must accurately reflect the true differences in the delivery devices. Many hospitals today are required to use labor statistics that come from outside our profession. This may reflect a weakness in the usefulness of the Uniform Reporting Manual.

Many factors have led to the confusion surrounding the selection of aerosol delivery devices. One example is the "breath-hold" requirement that has "found" its way into many policies and protocols that are used throughout our profession. The "breath-hold" requires that a patient must be able to hold his or her breath for a specific period of time in order to perform a pMDI maneuver effectively. However, this requirement has never been quantified and has absolutely no scientific basis for its use.[8] Interestingly, requirements of this type are often used to determine which delivery device is selected! As a profession, we must embrace the most current data regardless of the outcome it may have on the revenue produced and/or personnel needed to accomplish our assigned tasks.

We must ask, "What drives our decisions as to which delivery device we will select?" It appears that in some situations it is not current research, but strange traditions or even fear that may be driving our decisions. One common example of such a tradition is when aerosolized bronchodilators are always given via nebulizer to mechanically ventilated patients in order that a therapist can do multiple procedures simultaneously, thereby handling a much higher volume of work with fewer people.[9] Though this reasoning may appear noble, it is a practice that may devalue the role of the therapist at the bedside into someone who can simply pour medicine into a cup, start the device, and then move on to the next patient. These types of clinical practices may be an answer as to why pMDIs and DPIs are underutilized in the United States.

As medications become more expensive and the deposition of precise amounts of aerosol more crucial, the question of what role the respiratory therapist will continue to play looms ominous.[10] In order for the respiratory care profession to remain the experts in the field of aerosol delivery, we must deal with the data we now have on hand in a positive and aggressive manner. Some clinical traditions will change, and hopefully respiratory care professionals will be leading the charge.

In conclusion, a profession that is not true to itself is a profession that will always fall short of its purpose. Several serious questions remain. Has self-preservation been a factor within our profession in the underutilization of pMDIs and DPIs? Have we failed to realize that we are bound by a statement of ethics and professional conduct that requires we do the most efficient procedure? (Statement of Ethics and Professional Conduct of the American Association for Respiratory Care, especially the following two statements: "Promote the positive evolution of the profession, and health care in general, through improvement of the access, efficacy, and cost of patient care." "Refrain from indiscriminate and unnecessary use of resources, both economic and natural, in their practice.")

Would the United States be noted for its underutilization of a device if that particular device required more labor on the part of respiratory therapists instead of less? Would the respiratory care profession be more aggressive in getting the reimbursement policies changed if the change resulted in an increased need for respiratory therapists instead of less? How can the respiratory care profession be the leader in educating other clinicians and the public if we are confused and divided among ourselves? It is time for the respiratory care profession to become the experts in the delivery of aerosols and lead everyone out of the fog.

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The Lack of Data on Off-Label Use of Noninvasive Positive Pressure Ventilation in Children

Children hospitalized for treatment of respiratory diseases such as obstructive sleep apnea, central hypoventilation, and chronic lung disease have been placed on noninvasive positive pressure ventilation (NPPV) as an alternative to invasive forms of ventilation.[1] The ultimate goal of improving alveolar ventilation has in most cases been met with NPPV and without the complications that can occur with invasive therapies.[2] The use of NPPV in children has grown over the years. No longer is this method confined to neonates in the hospital. NPPV can be seen used on children in various sites, such as rehabilitation centers, long-term medical facilities, and the home setting. This is a problem! These practices are off-label uses of a product.[3]

Although NPPV has become widely used, very little in the way of research has been done in this area of respiratory care. Almost no research or discussion can be found for those of us who need guidance.[2] As a respiratory therapist for over 25 years, I have been frustrated in my desire to gain more knowledge regarding this relatively new form of ventilation. The question I present is: Why has this topic been avoided?

One of the reasons that might deter health care professionals from using NPPV could be that NPPV has not been approved for use in children.[4] The Food and Drug Administration and companies that manufacture flow generators for NPPV[5] do not recommend the use of devices that provide NPPV in children under 30 kg.[2] This off-label use of NPPV equipment needs to be investigated for all concerned.

Surprisingly few medications or medical devices for children have actually been studied for pediatric use. When treatments are approved for adult use, such as medications, the effect on children is rarely investigated.[6] This is not to say that effective treatment with NPPV in children is unseen, but that studies should be done on possible hazards.

The most widely used aerosolized bronchodilator, albuterol, contains a labeling disclaimer for use in children,[7] yet it is hard to deny its effectiveness in the pediatric population. Should we continue in the same path and let time determine the safety of NPPV? When a physician prescribes a drug for an unapproved use, that physician should make a diligent effort to become aware of the facts and be convinced that the benefits outweigh the possible risks.[8] If this is true, then why are we blindly using bi-level pressures on children without adequate research?

So where does that leave a respiratory therapist when presented with the dilemma of applying unapproved devices? First, equipment must be modified, parameters set, the child's condition assessed, and the device attached to the patient, all without manufacturer guidelines.

In the past, the FDA restricted dissemination of information and studies done by medical manufacturers on the off-label use of drugs, devices, or biologics.[9] On July 28, 1999, Judge Lamberth of the United States District Court for the District of Columbia issued a decision in the Washington versus Henning litigation regarding off-label use: the federal court ruling found that it was unconstitutional for the Food and Drug Administration to have restrictions on the ability of manufacturers to disseminate truthful information to medical professionals on off-label use of a product. The court also found that content or speakers to an independent program or providers in connection with a medical education seminar or symposium shall not be restricted, sanctioned, or limited.

With these changes it should be easier to obtain data for pediatric NPPV use, but sadly the discussion is not forthcoming. With all this said, why has the health care community failed to study the use of NPPV, especially in the home, where most of the safeguards, such as alarms, 24-hour medical care, and monitoring as done in the hospital setting, are absent.

There are some potential hazards during pediatric ventilation with NPPV. Inadvertent increase in airway pressure can be caused by electrical power surge to the unit, which can increase the pressure from 5 cm H_2O to 35 cm H_2O![4] This sudden change in pressure can cause pneumothorax in children. Gastric distention has been seen in patients using NPPV flow generators, which in turn may cause aspiration.[1] There have been many other complications in children and adults, but not enough data are in to attribute them to NPPV alone.

The medical complications possible with NPPV, the devices' limitations, and the liability issues have not been addressed. At a time when more pediatric patients are treated with NPPV, more information is greatly needed.

I challenge all health care personnel to attack the lack of guidelines concerning the use of NPPV in children, wherever it is used. We must begin studying this topic as quickly as possible.

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Fraser and Paré's Diagnosis of Diseases of the Chest. 4th edition (4-volume set). RS Fraser MD, N Colman MD, NL Müller MD PhD, PD Paré MD. Philadelphia: WB Saunders. 1999. Hardcover, illustrated, 3,190 pages plus index, $395.

The reader interested in pulmonary diseases can choose from among several textbooks in today's medical section of bookstores. The well-known textbook by Fraser and Paré, **Diagnosis of Diseases of the Chest**, recently came out with a new, fourth edition that is certainly worth a look.

The fourth edition again spans 4 volumes. Volume One describes the normal chest, investigative methods and radiologic signs of chest diseases, and developmental lung diseases. It also includes a commented glossary of terms commonly used in chest medicine. Volume Two reviews pulmonary infections and pulmonary neoplasms. Volume Three discusses immunologic lung diseases, embolic lung diseases, pulmonary hypertension and edema, and diseases of the airways. Finally, Volume Four presents information about inhalation/aspiration of particulates, solids and liquids, diseases caused by toxins, drugs, and irradiation, traumatic chest diseases, metabolic pulmonary diseases, pleural diseases, mediastinal diseases, diseases of the diaphragm and chest wall, and pulmonary diseases associated with a normal chest radiograph.

Each chapter is preceded by brief tables of contents, with reference to the relevant page—a very helpful feature. In general, the discussion of specific diseases is consistently subdivided into epidemiology, etiology and pathogenesis, pathology characteristics, radiologic manifestations, clinical manifestations, laboratory findings, prognosis, and natural history. This allows the reader to quickly orientate within chapters. Among those subdivisions, the 4 authors emphasize descriptions of radiologic findings and clinical history and thoroughly review and reference literature available on each topic. They support these by discussion of etiology and pathogenesis, while humbly acknowledging that the rapid increase in research findings, especially regarding cellular and molecular mechanisms, makes it difficult for only 4 authors, three of whom had only limited involvement in fundamental research, to timely and adequately present new developments. Interestingly, the authors decided not to discuss treatment options, which the reader might find rather surprising, given the size of this work.

The text is clearly written and well organized. One of the major assets is the wealth of high-quality radiographic images, including chest radiographs, computed tomography images, and magnetic resonance images. Given the impressive improvements in computed tomography, including high-resolution imaging and spiral computed tomography, the authors added numerous new illustrations of the normal lung and pathology findings. The reader will find detailed black and white macroscopic and microscopic illustrations of the various diseases of the chest. In this fourth edition, the authors also supplemented new sections on lung transplantation, human immunodeficiency virus infection, pulmonary hemorrhage syndromes, and pulmonary neoplasia.

Overall, the 4 volumes appear in an elegant green cover and are still comfortable to handle. Each volume contains a list of contents and a thorough index across all 4 volumes. To avoid having to look in a different volume, the authors allowed occasional repetitions. I encountered only very few typographical or printing errors. Sometimes the extensive lists of references after each chapter contained avoidable repeats of the same reference.

The authors state that they would like to provide a concept of diseases of the chest and an approach to diagnosis. Following a traditional teaching method, the chapters discuss disease entities rather than symptoms or findings. To aid the reader in a differential diagnostic approach, the appendix in Volume Four contains 17 "tables of differential diagnosis." Compared to prior editions, these tables were simplified and focus on more frequently encountered conditions. To make the tables more readable, clinical and laboratory findings were deleted and the table format was changed from vertical to horizontal orientation. Again, thoughtfully selected radiographs help clarify the various categories of radiographic findings.

The work is intended primarily for specialists in chest disease, including pulmonologists, thoracic surgeons, chest radiologists, and pathologists, and also interested residents in training. The 4 volumes serve as a detailed, well referenced, and readable textbook for anyone interested in in-depth study of diseases of the chest. Important historical and recent (until 1997) references are a helpful guide to review disease categories.

However, there are a few points worth criticizing. First, the authors describe in a very detailed way various findings in the literature about each of the chest diseases. It would be helpful to include more summary tables or algorithms in these chapters to aid the clinician in day-by-day decision-making and to emphasize important points. Second, a major drawback seems to be the lack of discussion of treatment options. Even if the practicing clinician will have to update him/herself about current recommendations of state-of-the-art treatment, it would be very interesting and helpful to find a thorough review of treatment options and their development over time included in this work. Furthermore, the book contains excellent black-and-white illustration, but no color illustrations; color would especially improve the histologic images. Moreover, in past editions, the tables of differential diagnosis also included more less-frequently-encountered diseases and helped the clinician in remembering those. Although it is extremely important to consider the prevalence of a disease in one's clinical and diagnostic approach, it would also be helpful to note uncommon diseases, which could escape our usual differential diagnosis, in a summary table. Diagnostic algorithms could help address this issue. For example, the authors opted to exclude the table about differential diagnosis of pleural effusions from the appendix, as this is based mostly on laboratory and not radiologic findings. This could have been added in the form of a diagnostic algorithm to the chapter on pleural effusions. In addition, the authors deleted references to pages in the text from the tables of differential diagnosis, which had in previous editions been helpful as quick guides to find the relevant chapter.

In summary, the fourth edition of **Diagnosis of Diseases of the Chest** is a very thorough, well referenced, and well written textbook about diseases of the chest, with exceptional black-and-white illustrations and helpful tables to aid in differential diagnosis. It will be a useful addition to any medical library and for anyone interested in a detailed review of diseases of the chest.

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Comprehensive Respiratory Medicine. Richard K Albert MD, Stephen G Spiro MD, James R Jett MD, Editors. London: Mosby, 1999. Hardcover, illustrated, 796 pages, $125.

Does the world need another textbook of respiratory medicine? Drs Albert, Spiro, and Jett think so. **Comprehensive Respiratory Medicine**, their contribution to this growing literature, was developed primarily to reflect the international scope of respiratory science. In doing so, it comprises the contributions of 120 authors from 10 countries. A secondary purpose was to take advantage of improved technology for the display of visual information, particularly in chest imaging. The result is a remarkably useful volume that should appeal to a wide audience.

The text is divided into 5 major sections. The first, devoted to structure and function, addresses the foundations of respiratory medicine. The 5 chapters in this section are heavily oriented towards imaging and indirectly address thoracic anatomy. The treatment of imaging techniques, particularly chest radiography and computed tomography, is outstanding. The format is a logical sequence of illustrated anatomy and radiographic principles leading to applications in specific disease states with dramatic images seldom seen outside of the radiologic literature. The section devoted to mediastinal anatomy very effectively couples mediastinal images with labeled illustrations to convey an understanding of this complex arrangement of intrathoracic structures. The presentation on high-resolution computed tomography of the lungs develops this subject by presenting the anatomy of the secondary pulmonary lobule, followed by a discussion of specific imaging patterns (eg, ground glass opacification) and their recognition and significance in specific clinical disorders. Chapters devoted to magnetic resonance imaging and radionuclide scanning are helpful but seem less focused. Magnetic resonance imaging is infrequently used in clinical thoracic imaging, and the chapter on lung scan techniques would have been enhanced by a discussion of the diagnosis of pulmonary embolism. The chapter on physiology is superb and concise, as is a section on host defense. The editors note that a comprehensive review of cell and molecular biology is not the purpose of this effort, and they successfully resist that temptation.

The second section is devoted to diagnostic and therapeutic procedures used in respiratory medicine and is one of the strengths of this text. The 5 chapters address common procedures such as flexible and rigid bronchoscopy, pleural biopsy, intrathoracic needle biopsy, tube thoracostomy, and mediastinoscopy/thoracoscopy. These chapters are particularly useful for physicians in training. The procedures are comprehensively discussed with a well referenced format of indications, techniques, and illustrated guidelines. Regrettably, procedures are rarely addressed in textbooks of medicine and these topics fill an important void found in most clinical respiratory textbooks.

The third section addresses principles of respiratory care. These 4 chapters address invasive and noninvasive ventilation, hemodynamic monitoring, and airway management. The section on invasive ventilation provides a broad overview of the physiologic principles in assisted ventilation, as well as their application and measurement. Practical protocols for the application of positive end-expiratory pressure and ventilator weaning are particularly useful. The chapters devoted to noninvasive ventilation, hemodynamic monitoring, and airway management have a practical focus, with helpful sections on techniques of arterial and central venous cannulation and endotracheal intubation. Illustrated guidelines also teach effective trouble-shooting techniques. Sections devoted to respiratory symptoms are concise and patient-focused. Each involve a brief review of pathophysiology coupled with an algorithmic clinical approach. A chapter in this section devoted to the history and physical examination of patients with respiratory symptoms is missing and would have been useful.

The remainder of the text is devoted to specific clinical entities. Some topics, such as respiratory infections and airways diseases, are presented in depth. Others, such as pulmonary vascular disease, pleural disease, and diffuse lung disease, receive less attention. The content of these chapters is generally well developed and current. Subjects such as the management of acquired immunodeficiency syndrome and implications for new therapies, such as highly active antiretroviral therapy, are brief but thorough. A discussion of airways diseases carefully reviews pharmacology, physiologic abnormalities of gas exchange, staging (including both European Respiratory Society and American Thoracic Society evaluation systems), and management. The chapters devoted to collagen vascular disorders are standard in their approach. Additional chapters discussing pregnancy, bone marrow transplantation, and pulmonary complications of hematologic, liver, and inflammatory bowel disorders provide useful clinical information for clinicians who are frequently consulted to evaluate and manage the respiratory complications of these diseases.

Comprehensive Respiratory Medicine derives material from a diverse group of clinicians and investigators. This clearly strengthens its value and presents a more balanced view of pulmonary medicine for those of us accustomed to a primarily North American perspective. The risk in this approach is the possibility of redundancy, disagreement, or error. The editors have done a superb job in this respect. While there are occasional disagreements (such as a difference in the portrayal of mediastinal compartments in Figures 1.20 and 74.1), important discrepancies are uncommon. There are also few typographical errors of consequence (one was detected in the title of a contributor who is editor in chief of this journal). The layout of the text is orderly, but I found the numerical system identifying sections and chapters confusing. The visual presentation is superb. While many textbooks present a bland, monochromatic and multivolume format, this refreshing single volume uses a sophisticated strategy of multicolor graphics, striking clinical photography, and concise summary tables and figures without sacrificing content.

This text is ideal for respiratory clinicians and fellowship trainees, and established clinicians and scientists may be surprised by the depth of the material and its clinical usefulness. The editors have established a resource with a well defined vision, and the distinguished group of contributors has adhered to it. Clinicians will not only want this volume in their library, they may find themselves referring to it more frequently than other more established respiratory textbooks.

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Textbook of Respiratory Medicine, 3rd edition (2-volume set). John F Murray MD DSc, Jay A Nadel MD DSc, Editors. Philadelphia: WB Saunders. 2000. Hardcover, illustrated, 2,562 pages plus appendix and index, $325.

Every discipline has its reference textbook, the gold standard tome that can be found on most bookshelves, the common starting place of inquiry. Murray and Nadel's **Textbook of Respiratory Medicine** has, for the most part, been such a resource for pulmonologists and others concerned with respiratory medicine for the last 12 years. Attempting to keep pace with the new discoveries and rapid advances in pulmonary medicine, a third edition of the textbook has been published, 6 years since the second edition was published in 1994. The new edition brings with it two new editors, 68 new authors, more than 2,500 pages, over 21,000 references, and a new green color for the two-volume set; it constitutes an update to the second edition rather than a more comprehensive overhaul.

The goal of the book, as stated by the authors in the preface, is to integrate and consolidate the expanding base of essential knowledge available in pulmonary medicine into a single comprehensive textbook. In so doing, the authors strive to integrate basic science—including anatomy, physiology, pharmacology, and pathology—with the clinical practice of respiratory medicine. Roughly 20% of the book (536 pages) is dedicated to the scientific principles of respiratory medicine, and the majority (2,023 pages) covers clinical pulmonary medicine. By nature of its content, comprehensiveness, and clinical emphasis, this text will appeal first and foremost as a definitive reference textbook to the chest physician, though others such as general physicians, medical trainees, nurses, and respiratory therapists may find it a useful (albeit probably more-than-is-needed) text as well.

In Part I, "Scientific Principles of Respiratory Medicine," the text is divided into 5 sections, titled "Anatomy and Development of the Respiratory Tract," "Respiratory Physiology," "Respiratory Pharmacology," "Defense Mechanisms and Immunology," and "Respiratory Pathology and Inflammation." Most of the 19 chapters contained within represent updates of their predecessors, some have been reorganized, and two new chapters, "Genetic Basis of Lung Disease" and "Pulmonary Surfactant," have been added. Generally, this part of the book succeeds in providing a fairly comprehensive review of the scientific principles underlying respiratory medicine. Material is presented in such a way as to be accessible to both scientist and clinician, and in most cases clinical relevance is well linked to its basic counterpart. Several chapters, however, such as "Genetic Basis of Lung Disease," "General Pharmacologic Principles," and "Monocytes, Macrophages, and Dendritic Cells of the Lung," are detailed and basic enough to be of more interest to the lung biologist than to the clinician.

Part II, "Manifestations and Diagnosis of Respiratory Disease," is composed of 11 chapters covering routine diagnostics used in evaluating patients with respiratory disease. It starts with a 3-chapter symptom-based section describing an approach to the main symptoms encountered in pulmonary medicine: dyspnea, cough, and chest pain. Following are 8 chapters that discuss radiographic techniques, microbiologic evaluation, pulmonary function testing, bronchoscopy, preoperative evaluation, and evaluation of respiratory impairment and disability.

Part III, "Clinical Respiratory Medicine," contains 68 chapters covering the wide spectrum of pulmonary disease, from infections, malignancy, obstructive lung disease, restrictive lung disease, disorders of the pulmonary circulation, interstitial lung disease, occupational lung disease, pleural and mediastinal disease, to disorders in the control of breathing, respiratory manifestations of extrapulmonary disorders, and respiratory failure. The overall organization of chapters has not changed substantially from the second edition, though a few chapters have been combined and consolidated, while others have been expanded. The material presented is clear and concise, yet thorough and easy to read. The tendency of physicians may be to dismiss textbooks as too basic and to look first to the literature for answers. I was pleasantly surprised by the amount of useful information present in this text, its scope and detail, and its reference to recent studies. When I had a specific clinical question, more often than not the question was answered, clinical knowledge was refreshed, or I was provided with several references for further study. The appearance of the tables, charts, and pictures has changed only slightly from the previous edition. Although not presented in color, illustrations remain clear and well reproduced.

Two challenges inherent in all reference textbooks include staying up to date with the ever-evolving literature and managing to cover specific topics in more than cursory detail. This textbook does well in both regards. Chapters are extensively referenced and include classic articles as well as recent publications, through 1999. Chapter 51, "Pulmonary Thromboembolism," is one example where the textbook keeps apace with recent advances in clinical pulmonary medicine. The new edition brings with it discussions of the role of activated resistance to protein C in thromboembolism, the utility of the D-dimer assay, and helical computed tomography in the diagnosis of pulmonary embolism, and updates on the surgical treatment of chronic thromboembolic pulmonary hypertension. In Chapter 57, "Idiopathic Interstitial Pneumonias," the authors again prove up to the task, completely reorganizing the chapter to present the most recent classification of interstitial lung diseases, including the entities of interstitial pulmonary fibrosis, usual interstitial pneumonia, desquamative interstitial pneumonitis, respiratory bronchiolitis-associated interstitial lung disease, lymphocytic interstitial pneumonitis, acute interstitial pneumonia, cryptogenic organizing pneumonia, and nonspecific interstitial pneumonitis. The text can by no means claim to be the most comprehensive authority for specific diseases, nor is this its goal. However, the book does succeed, I believe, in providing the most comprehensive and well-cited reference textbook of pulmonary medicine available.

That being said, let us examine two other pulmonary textbooks of similar proportion. Fishman's third edition of *Pulmonary Diseases and Disorders*, published in 1998, is another reference pulmonary textbook with a similar format. Comparing 3 chapters in detail and scanning many others, the two were closely matched in organization and content, though I found the **Textbook of Respiratory Medicine** to be a bit more detailed and data-driven, and certainly more extensively referenced. Though the color images in Fishman's book are nice, information was presented in a more general fashion. Fraser and Pare's *Diagnosis of Diseases of the Chest*, a 4-volume set, provides a more in-depth study of the diagnosis of pulmonary disease, with a clear radiologic emphasis, packed full of beautiful images. It does not include any discussion of therapeutics, however. Of the three, the latest edition of the **Textbook of Respiratory Medicine** can truly claim to be a gold standard in its field. It is comprehensive, well researched, well written, well illustrated, and nicely presented. It is a necessity for any pulmonologist, a valuable resource for department or medical school libraries, and a wealth of information for others involved in the clinical care of patients with pulmonary diseases.

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Textbook of Critical Care, 4th edition. Ake Grenvik MD PhD, Senior Editor; Stephen M Ayres MD, Peter R Holbrook MD, and William C Shoemaker MD, Editors. Philadelphia: WB Saunders. 2000. Hardcover, illustrated, 2,227 pages, $165.

This is a comprehensive textbook of critical care medicine with two great areas of strength: trauma resuscitation and pulmonary medicine, including mechanical ventilation. Physicians in the fields of critical care, surgery, and emergency medicine are the primary audience, but the 440-page pulmonary section—one fifth of the book—will be of interest to many respiratory therapists as well. Nurses in critical care or emergency departments may find the section on organization of a critical care service of particular interest.

The single large volume is attractively typeset. Illustrations are clear and are professionally done: no hastily doodled computer graphics here. Typographical errors are likewise scarce, though a few did escape the editors' eyes: one chapter describes plasma volume as "4 mL/kg": the value should be 40. References are generally current and relevant. The index is comprehensive and helpful.

This massive book is intended to move smoothly from initial resuscitation through trauma to organ-specific critical care issues. The first section, "Resuscitation and Medical Emergencies," is the core of the book, beginning with cardiopulmonary resuscitation (CPR)/advanced cardiac life support and covering exsanguination, shock, drug overdoses, drowning, and acute coronary syndromes—in short, the pre-intensive-care-unit care of the critically ill patient. There are separate sections on transplantation, on organization of critical care, and on ethical decision-making. Sections are divided into chapters, each individually tailored to a single issue or small focus; there are over 200 chapters in all, divided into 15 sections.

This text, almost uniquely in an era of evidence-based medicine, is built around the unifying *idea* of oxygen delivery, the foremost proponent of which was and remains Dr William Shoemaker. The chapters on shock, invasive and noninvasive monitoring, and resuscitation algorithms are his. He invokes the framework of oxygen delivery in explaining the pathophysiology of acute respiratory distress syndrome—a controversial position at best, but one that he defends vigorously. After 16 years and 4 editions, this remains *Shoemaker's* textbook of critical care.

The structure of the book, with its large sections and bite-sized chapters, results in some unevenness of tone. For example, in Chapter 1, "Cardiopulmonary-Cerebral Resuscitation," page 15 dwells on the inadequacy of current advanced cardiac life support protocols and advocates that "open-chest CPR . . . be taught again to all physicians and medical students." The chapter on pediatric resuscitation recommends only closed-chest CPR and a round or two of epinephrine, then terminating efforts in order to avoid saving a severely brain-damaged child—the difference in outlook is striking.

Likewise, the currency of some chapters is questionable. Chapter 6, "Intravenous and Intra-arterial Access," is replete with illustrations of ways to obtain access—all of which display the catheter-through-needle technique, which has been superseded by the Seldinger (catheter over wire) technique in almost every application, eliminating the risk of sheared-off catheters. The authors of this chapter favor the use of multiple single-lumen central lines—with multiple venipunctures—rather than the commonly used multiple-lumen catheter. There is no mention of the use of ultrasound guidance for central line placement. This chapter deserves a complete revision—fast-forward 20 years.

The section on respiratory function and mechanical ventilation, by contrast, is a gem. It proceeds in logical, coherent fashion from the fundamentals of respiratory mechanics and gas flow through airway access and conventional modes of mechanical ventilation to new strategies such as partial liquid ventilation. The strategy of small, tightly focused chapters works well here. For example, the chapter on independent lung ventilation concisely and helpfully describes the techniques required to perform whole lung lavage (usually done for pulmonary alveolar proteinosis) in enough detail to allow an operator to proceed without further reference. Several chapters touch on acute respiratory distress syndrome, as might be expected. Some focus on the now-conventional practice of limiting transpulmonary pressures, and, by implication, tidal volume; others explore such options as high-frequency ventilation and continuous gas insufflation. Adjunctive strategies such as nitric oxide and prostacyclin also receive due attention. Robert Kacmarek's chapter on controlled mechanical ventilation is a joy, a mini-seminar on a wide range of relevant topics: the effects of inverse-ratio ventilation, the management of intrinsic positive end-expiratory pressure, the true significance of inflection points in the pressure-volume curve, and even the high rate of ventilator false alarms.

Weaning from mechanical ventilation is covered in 4 separate chapters, each written by a giant in the field. Neil MacIntyre reviews patient-ventilator interactions as well as weaning in hypoxemic respiratory failure. John Marini outlines partial ventilatory assist—a closely related concept. Martin Tobin's name headlines the chapter on weaning in obstructive airway disease. Splitting this topic among lesser authors would have led to redundant rehashes of "conventional wisdom." With authors of this caliber, however, the depth and perspective thus gained are worth the overlap.

New to this edition are several chapters on the latest developments in cellular biology. The roles of cytokines, nitric oxide, and other intracellular and intercellular signals are explored in detail. Two chapters on apoptosis, one focused on bench research and one on clinical applications, round out this exploration of the frontiers of critical care—the search for new, genetically-derived therapies to halt or reverse the processes of cellular injury. It is remarkable to see such cutting-edge material in a textbook intended for a general audience, but it is entirely in concordance with the spirit of the book.

This is a textbook inspired by pioneers and visionaries—men and women who laid the foundations of critical care. It is not the first textbook I would recommend to a newcomer to the field—someone looking for an easily assimilated overview of a large subject. There are weak chapters and duplications, controversial ideas put forward as proposed standards, conflicting approaches to the same problems, and chapters devoted to bench research that may never see clinical application. This book is not so much "state of the art" as "state of the dream." There are many "dreams" in this compilation: a combined emergency medicine-critical care service, a biochemical pathway to block cell death after severe injury, a ventilation strategy optimizable for any respiratory condition, and too many others to enumerate. I recommend this volume to the seasoned practitioner looking for new insights, new techniques, or just a glimpse of what may be around the corner in critical care.

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Clinical Practice in Respiratory Care. James B Fink MS RRT and Gerald E Hunt MS LVN RRT RPFT, Editors. Philadelphia: Lippincott Williams & Wilkins. 1999. Hardcover, illustrated, 516 pages, $52.

This new book has 15 contributing authors and consists of 18 chapters, ranging from "The Role of the Respiratory Care Practitioner" (Chapter 1) to "Home Respiratory Care" (Chapter 18). Since this book deals with clinical practice in respiratory care, a review can best be discussed by dividing the chapters into 3 types of learning material: background information leading to clinical practice, basic respiratory care procedures, and advanced respiratory care procedures.

In addition to Chapter 1, three other chapters make up the background information leading to clinical practice. These are Chapter 2, "Legal and Ethical Considerations," Chapter 3, "Respiratory Anatomy and Physiology," and Chapter 5, "Respiratory Pathophysiology."

Basic respiratory care procedures are covered by 10 chapters: Chapter 4, "Diagnostic Procedures at the Bedside"; Chapter 6, "Assessing Signs and Symptoms of Respiratory Dysfunction"; Chapter 7, "Physical Properties of Gases and Fluids"; Chapter 8, "Pharmacology Associated with Respiratory Care"; Chapter 9, "Infection Control and Safety"; Chapter 10, "Gas Therapy"; Chapter 11, "Humidity"; Chapter 12, "Aerosol Drug Therapy"; Chapter 13, "Bronchial Hygiene and Lung Expansion"; and Chapter 14, "Airway Care."

Four chapters cover the respiratory care procedures that generally require more training and extensive clinical experience. They are Chapter 15, "Mechanical Ventilation"; Chapter 16, "Pediatric Considerations"; Chapter 17, "Subacute Care"; and Chapter 18, "Home Respiratory Care."

Since this book devotes only 4 chapters to advanced respiratory care procedures, the overall content of the book is more useful as an introductory text for beginning respiratory therapists or as a reference source for experienced respiratory care practitioners. In the past decade, the scope of practice in respiratory care has become much broader, and this trend will probably continue. A future edition should include more critical care topics such as noninvasive cardiology, cardiovascular drugs, advanced cardiac life support protocols, and hemodynamic monitoring.

The overall layout of the book is good but lacks consistency or clarity in some cases. For example, some chapters use references (eg, Chapters 1, 8, and 9), whereas others use bibliography (eg, Chapters 2 through 7). In another example, some case scenarios are presented with answers (eg, page 104), whereas others are without answers (eg, page 145). The use of boxed features to highlight important concepts is nicely done in most cases but lacks effectiveness in other cases. For instance, the "critical thinking challenges—shifting care plans" feature on page 80 simply provides a list of technology options in spirometry; this does not provide leading questions or information leading to the use of critical thinking skills.

Topics in this book are adequately presented. However, the title for Chapter 16 should be "Neonatal and Pediatric Considerations," since the chapter deals almost entirely with neonatal respiratory care.

The major shortcoming of the book is the lack of accuracy on some key topics in the practice of respiratory care. Figure 10–5 on page 252 and Figure 10–6 on page 253 show right-to-left shunt; yet the figure legends describe the illustrations as left-to-right shunt. The text on page 251 also fails to clarify those two types of shunt.

On page 418, under "indications and recommendations," the text describes that the "minimal levels of 5 to 10 cm H_2O of pressure support should always be used . . . ," but it does not provide the titration end points.

On page 420, under the "critical thinking challenges" and "case scenarios" on the topic of positive end-expiratory pressure (PEEP), the example describes using 12 cm H_2O of applied PEEP to counter the effect of 12 cm H_2O of intrinsic PEEP. That case scenario would be a good place to challenge the readers on the application of applied PEEP—one not to exceed 85% of intrinsic PEEP. Unfortunately, this "critical thinking challenge" not only fails to challenge the readers, it provides an answer that is not supported by published studies.

Under "rapid shallow breathing index" on page 424, the text reads ". . . measure the patient's respiratory rate and average tidal volume from the ventilator." This is a major oversight, as later in this section it describes the average tidal volume from the patient.

There are other minor errors. For example, the alveolar partial pressure of oxygen in Figure 3–27 on page 62 should be 100 mm Hg instead of 40 mm Hg. And on page 93, the measurements extrapolated from the curves in Figure 4–21 do not show pre-FEV_1 (forced expiratory volume in the first second) of 2.71 L and post-FEV_1 of 3.07. On page 94, the flow-volume curves in Figure 4–25 do not show time markings in seconds. The readers may not realize that the arrows are pointing at the pre-bronchodilator and post-bronchodilator FEV_1 measurements.

In spite of the aforementioned deficiencies, the overall coverage and presentation make this book a nice text for respiratory therapy students. Instructors should read, uncover, and point out the text's errors to students so that they may benefit from the book with minimal confusion and avoid misunderstanding some important concepts in the practice of respiratory care.

There is a lighter side to this text as well. In the preface, the editors wrote "We have attempted to invoke a casual style [and] humor . . . ." The illustrations of a blue bloater and a pink puffer on page 140 showthat their eyes have been blocked as if an attempt was made to conceal their identities! In summary, **Clinical Practice in Respiratory Care** is easy to read and it covers all essential topics on respiratory care with sufficient depth and detail. With more accurate proofreading in the next edition, it should be even more useful as a text for beginning students and as a reference source for RTs.

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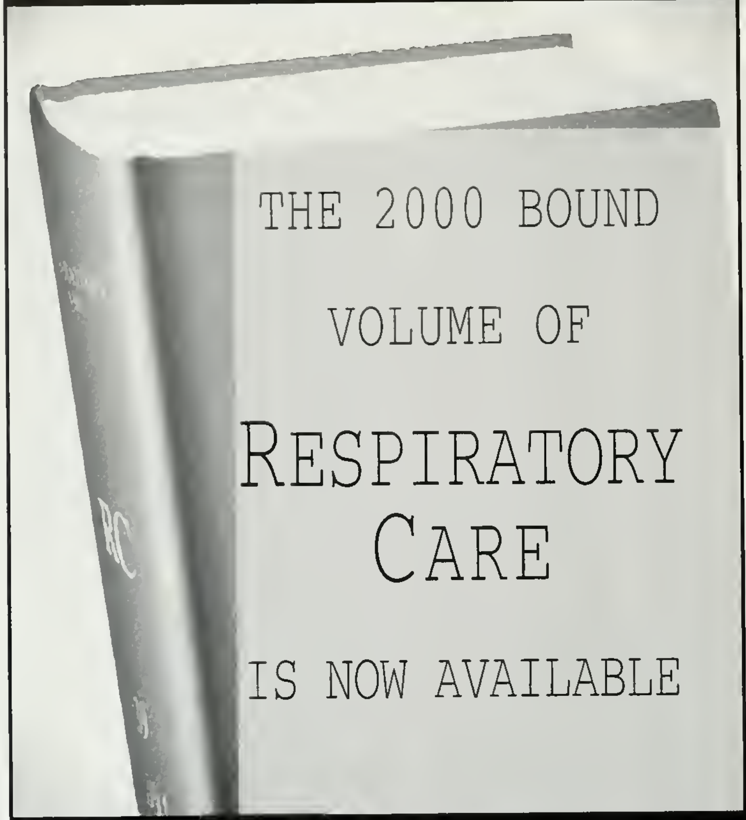

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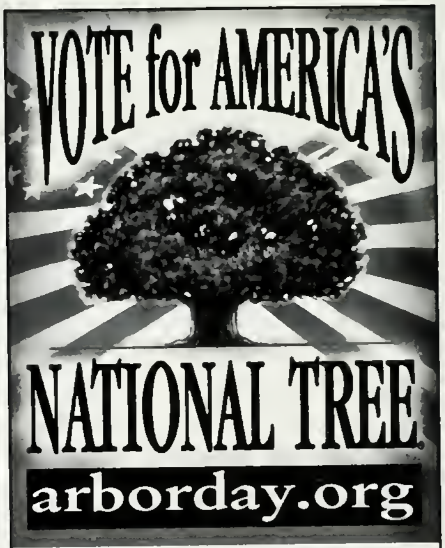

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The American Association for Respiratory Care and its science journal, RESPIRATORY CARE, invite submission of brief abstracts related to any aspect of cardiorespiratory care. The abstracts will be reviewed, and selected authors will be invited to present posters at the OPEN FORUM during the AARC International Respiratory Congress in San Antonio, Texas, December 1-4, 2001. Accepted abstracts will be published in the October 2001 issue of RESPIRATORY CARE. Membership in the AARC is not required for participation. All accepted abstracts are automatically considered for ARCF research grants.

SPECIFICATIONS—READ CAREFULLY!

An abstract may report **(1) an original study, (2) the evaluation of a method, device or protocol, or (3) a case or case series.** Topics may be aspects of adult acute care, continuing care/rehabilitation, perinatology/pediatrics, cardiopulmonary technology, or health care delivery. The abstract may have been presented previously at a local or regional—but not national—meeting and should not have been published previously in a national journal. The abstract will be the only evidence by which the reviewers can decide whether the author should be invited to present a poster at the OPEN FORUM. Therefore, *the abstract must provide all important data, findings, and conclusions.* Give specific information. Do not write such general statements as "Results will be presented" or "Significance will be discussed."

ESSENTIAL CONTENT ELEMENTS

Original study. Abstract *must* include (1) Background: statement of research problem, question, or hypothesis; (2) Method: description of research design and conduct in sufficient detail to permit judgment of validity; (3) Results: statement of research findings with quantitative data and statistical analysis; (4) Conclusions: interpretation of the meaning of the results.

Method, device, or protocal valuation. Abstract *must* include (1) Background: identification of the method, device, or protocol and its intended function; (2) Method: description of the evaluation in sufficient detail to permit judgment of its objectivity and validity; (3) Results: findings of the evaluation; (4) Experience: summary of the author's practical experience or a lack of experience; (5) Conclusions: interpretation of the evaluation and experience. Cost comparisons should be included where possible and appropriate.

Case report. Abstract *must* report a case that is uncommon or of exceptional educational value and must include (1) Introduction: relevant basic information important to understanding the case. (2) Case Summary: patient data and response, details of interventions. (3) Discussion: content should reflect results of literature review. The author(s) should have been actively involved in the case and a case-managing physician must be a co-author or must approve the report.

FORMAT AND TYPING INSTRUCTIONS

Accepted abstracts will be photographed and reduced by 40%; therefore, the size of the original text should be *at least* 10 points. A font like Helvetica or Times makes the clearest reproduction. The first line of the abstract should be the title in all capital letters. Title should explain content. Follow title with names of all authors (including credentials), institution(s), and location; underline presenter's name. Type or electronically print the abstract *single spaced in one paragraph on a clean sheet of paper, using margins set so that the abstract will fit into a box no bigger than 18.8 cm (7.4") by 13.9 cm (5.5"), as shown on the reverse of this page.* Insert only one letter space between sentences. Text submission on diskette is allowed but must be accompanied by a hard copy. *Data may be submitted in table form, and simple figures may be included provided they fit within the space allotted. No figure, illustration, or table is to be attached to the abstract form.* Provide all author information requested. Standard abbreviations may be employed without explanation; new or infrequently used abbreviations should be spelled out on first use. Any recurring phrase or expression may be abbreviated, if it is first explained. Check the abstract for (1) errors in spelling, grammar, facts, and figures; (2) clarity of language; and (3) conformance to these specifications. An abstract not prepared as requested may not be reviewed. Questions about abstract preparation may be telephoned to Linda Barcus at (972) 406-4667.

Early Deadline Allowing Revision. Authors may choose to submit abstracts early. Abstracts postmarked by May 31, 2001 will be reviewed and the authors notified *by letter only* to be mailed by June 15, 2001. Rejected abstracts will be accompanied by a written critique that should, in many cases, enable authors to revise their abstracts and resubmit them by the Final Deadline (July 17, 2001).

Final Deadline. The mandatory Final Deadline is July 17, 2001 (postmark). Authors will be notified of acceptance or rejection *by letter only*. These letters will be mailed by September 1, 2001.

Mailing Instructions. Mail (Do not fax!) 2 clear copies of the completed abstract form, diskette (if possible), and a stamped, self-addressed postcard (for notice of receipt) to:

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RESPIRATORY CARE

Manuscript Preparation Guide

RESPIRATORY CARE welcomes original manuscripts related to the science and technology of respiratory care and prepared according to the following instructions and the *Uniform Requirements for Manuscripts Submitted to Biomedical Journals* (available at http://www.acponline.org/journals/resource/unifreqr.htm). Manuscripts are blinded and reviewed by professionals who are experts in their fields. Authors are responsible for obtaining written permission to publish previously–published figures and tables from the original copyright holder. Accepted manuscripts are copyedited for clarity, concision, and consistency with RESPIRATORY CARE format. Before publication, authors receive page proofs for minor correction. Published papers are copyrighted by Daedalus Inc and may not be published elsewhere without permission. Editorial consultation is available at any stage of planning or writing for any submission: contact the Editorial Office.

Categories of Articles

Research Article: A report of an original investigation (a study). Must include Title Page, Abstract, Key Words, Background, Methods, Results, Discussion, Conclusions, and References. May also include Tables, Figures (if so, must include Figure Legends), Acknowledgments, and Appendices.

Review Article: A comprehensive, critical review of the literature and state-of-the-art summary of a topic that has been the subject of at least 40 published research articles. Must include: Title Page, Outline, Key Words, Introduction, Review of the Literature, Summary, and References. May also include: Tables, Figures (if so, must include Figure Legends), and Acknowledgments.

Overview: A critical review of a pertinent topic that has fewer than 40 published research articles. Same structure as Review Article.

Update: A report of subsequent developments in a topic that has been critically reviewed in RESPIRATORY CARE or elsewhere. Same structure as a Review Article.

Special Article: A pertinent paper not fitting one of the other categories. Consult with the Editor before writing or submitting such a paper.

Editorial: A paper addressing an issue in the practice or administration of respiratory care. It may present an opposing opinion, clarify a position, or bring a problem into focus.

Letter: A brief, signed communication responding to an item published in RESPIRATORY CARE or about other pertinent topics. Tables, Figures, and References may be included. The letter should be marked "For Publication."

Case Report: Report of an uncommon clinical case or a new or improved method of management or treatment. A case-managing physician must either be an author or furnish a letter approving the manuscript. Must include: Title Page, Abstract, Introduction, Case Summary, Discussion, and References. May also include: Tables, Figures (if so, must include Figure Legends), and Acknowledgments.

Point-of-View Paper: A paper expressing personal but substantiated opinions on a pertinent topic. Must include: Title Page, Text, and References. May also include Tables and Figures (if so, must include Figure Legends).

Drug Capsule: A miniature review paper about a drug or class of drugs that includes discussions of pharmacology, pharmacokinetics, or pharmacotherapy.

Graphics Corner: A brief case report discussing and illustrating waveforms for monitoring or diagnosis. Should include Questions, Answers, and Discussion sections.

Kittredge's Corner: A brief description of the operation of respiratory care equipment. Should include information from manufacturers and editorial comments and suggestions.

PFT Corner: A brief, instructive case report including pulmonary function testing, accompanied by a review of the relevant physiology and appropriate references to the literature.

Test Your Radiologic Skill: A brief, instructive case report involving pulmonary medicine radiography and including one or more radiographs. May involve imaging techniques other than conventional chest radiography.

Review of a Book, Film, Tape, or Software: A balanced, critical review of a recent release. RESPIRATORY CARE does not accept unsolicited book reviews; please contact the Editor if you have a suggestion for a book review.

Preparing the Manuscript

Print on one side of white 8.5 × 11 inch paper, with margins of at least 1 inch on all sides. Double-space the text and number the pages. Do *not* include author names, author institutional affiliations, or allusions to institutional affiliations anywhere except on the title page. On the Abstract page include the title but do not include author names. Begin each of the following on a new page: Title Page, Abstract, Text, Acknowledgments, References, each Table, each Figure, and each Appendix. Use standard English in the first person and active voice. Type all headings in initial-capital letters (eg, Background, Methods, Patients, Equipment, Statistical Analysis, Results, Discussion). Center the main section headings and place second-level headings on the left margin.

Abstract. Please ensure that the abstract does not contain any facts or conclusions that do not also appear in the body text. Limit the abstract to no more than 400 words.

Key Words. Research, Review, Overview, and Special Articles require Key Words. On the Abstract or Outline page, include a list of 6 to 10 key words or two-word phrases.

References. Assign reference numbers in the order that articles are cited in your manuscript. At the end of your manuscript, list the cited works in numerical order. Abbreviate journal names as in *Index Medicus*. List all authors. The following examples show RESPIRATORY CARE's style for references.

Article in a journal carrying pagination throughout the volume:

> Rau JL, Harwood RJ. Comparison of nebulizer delivery methods through a neonatal endotracheal tube: a bench study. Respir Care 1992;37(11):1233-1240.

Article in a publication that numbers each issue beginning with Page 1:

> Bunch D. Establishing a national database for home care. AARC Times 1991;15(Mar):61,62,64.

Corporate author journal article:

> American Association for Respiratory Care. Criteria for establishing units for chronic ventilator-dependent patients in hospitals. Respir Care 1988;33(11):1044-1046.

Article in journal supplement: (Journals differ in numbering and identifying supplements. Supply information sufficient to allow retrieval.)

> Reynolds HY. Idiopathic interstitial pulmonary fibrosis. Chest 1986; 89(3 Suppl):139S-143S.

Abstract in journal: (Abstracts citations are to be avoided, and those more than 3 years old should not be cited.)

> Stevens DP. Scavenging ribavirin from an oxygen hood to reduce environmental exposure (abstract). Respir Care 1990;35(11): 1087-1088.

Editorial in a journal:

> Enright P. Can we relax during spirometry? (editorial). Am Rev Respir Dis 1993;148(2):274.

Editorial with no author given:

> Negative-pressure ventilation for chronic obstructive pulmonary disease (editorial). Lancet 1992:340(8833):1440-1441.

Letter in journal:

> Aelony Y. Ethnic norms for pulmonary function tests (letter). Chest 1991;99(4):1051.

Corporate author book:

> American Medical Association Department of Drugs. AMA drug evaluations, 3rd ed. Littleton CO: Publishing Sciences Group; 1977.

Book: (For any book, specific pages should be cited whenever reference is made to specific statements or other content.)

> DeRemee RA. Clinical profiles of diffuse interstitial pulmonary disease. New York: Futura; 1990:76-85.

Chapter in book with editor(s):

> Pierce AK. Acute respiratory failure. In: Guenter CA, Welch MH, editors. Pulmonary medicine. Philadelphia: JB Lippincott; 1977:26-42.

Paper accepted but not yet published:

> Hess D. New therapies for asthma. Respir Care (year, in press).

Personal communication of unpublished data not yet accepted for publication: You must obtain written permission to cite unpublished data received via personal communication. Do not number such references, but instead make parenthetical reference in the body text of your manuscript. Example: "Recently, Jones found this treatment effective in 45 of 83 patients (Jones HI, University of the Cascades, 1999, personal communication)."

Tables. Tables should be consecutively numbered. Start each table on a separate page. Number and title the table and give each column a brief heading. Place explanations in footnotes, including all nonstandard abbreviations and symbols. Key the footnotes with the following symbols, superscripted, in the table body, and in the following order: *, †, ‡, §, ||, ¶, **, ††. Do not use horizontal or vertical rules or borders. Do not submit tables as photographs, reduced in size, or on oversize paper.

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Drugs. Precisely identify all drugs and chemicals used, giving generic names, doses, and methods of administration. Brand names may be given in parentheses after generic names.

Commercial Products. In the text, parenthetically identify commercial products only on first mention, giving the manufacturer's name, city, and state or country. Example: "We performed spirometry (1085 System, Medical Graphics, Minneapolis, Minnesota)." Provide model numbers if available, and manufacturer's suggested price, if the study has cost implications.

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Ethics. When reporting experiments on human subjects, indicate that procedures were conducted in accordance with the ethical standards of the *World Medical Association Declaration of Helsinki* (see Respir Care 1997;42(6):635-636) or of the institution's committee on human experimentation. State that informed consent was obtained. Do not use patient's names, initials, or hospital numbers in text or illustrations. When reporting experiments on animals, indicate that the institution's policy, a national guideline, or a law on the care and use of laboratory animals was followed.

Statistics. Identify the statistical tests used in analyzing the data and give the prospectively determined level of significance in the Methods section. Report actual p values in the Results section. Cite only textbook and published article references to support choices of tests. As with commercial products (see above), parenthetically identify any general-use or commercial computer programs used.

Units of Measurement. Express measurements of length, height, weight, and volume in metric units appropriately abbreviated; temperatures in degrees Celsius; and blood pressures in millimeters of mercury (mm Hg). Report hematologic and clinical-chemistry measurements in conventional metric and in SI (*Système Internationale*) units (units and conversion factors listed at Respir Care 1997;42(6):640). Show gas pressures (including blood gas tensions) in millimeters of mercury (mm Hg).

Conflict of Interest. On the cover page, authors must disclose any liaison or financial arrangement they have with a manufacturer or distributor whose product is addressed in the manuscript or with the manufacturer or distributor of a competing product. Such arrangements do not disqualify a paper from consideration and are not disclosed to reviewers. Reviewers are screened for possible conflict of interest.

Abbreviations and Symbols. Use standard abbreviations and symbols, listed at Respir Care 1997;42(6):637-642. Do not create new abbreviations. Do not use abbreviations in the title, in section headings, and do not use unusual abbreviations in the abstract. Use an abbreviation only if the term occurs 4 or more times in the paper. Define all abbreviations (ie, write out the full term on first mention, followed by the abbreviation in parentheses) and thereafter use only the abbreviation. Standard units of measurement and scientific terms can be abbreviated without explanation (eg, L/min, mm Hg, pH, O_2). Please use the following forms: cm H_2O (not cmH_2O), f (not bpm), L (not l), L/min (not LPM, l/min, or lpm), mL (not ml), mm Hg (not mmHg), pH (not Ph or PH), p > 0.001 (not p>0.001), s (not sec), S_{pO_2} (arterial oxygen saturation measured via pulse-oximetry).

Prior and Duplicate Publication. In general, do not submit work that has been published or accepted elsewhere, though in special instances the Editor may consider such material if the original publisher grants permission. Please consult the Editor before submitting such work.

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Submit three printed copies and one (3.5-inch) computer diskette. The printed copies should each include photocopies of all of the Figures, Tables, and Appendixes. On the diskette, the manuscript should be in one file and the tables in a separate file. If soft copies of the figures are available, they should also be in a separate file. *However, do not create scanned versions of figures borrowed from other publications; clear photocopies are preferable.* Include the completed Cover Letter and Checklist (see next page) and permission letters. Mail to RESPIRATORY CARE, 600 Ninth Avenue, Suite 702, Seattle WA 98104. Do not fax manuscripts. Receipt will be acknowledged.

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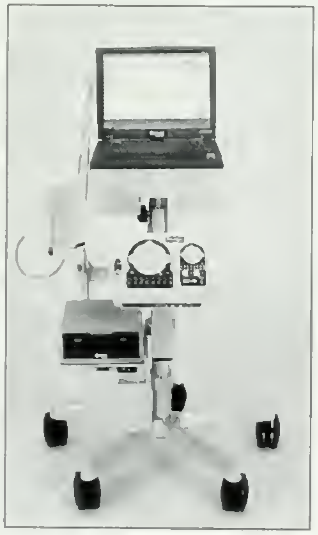
New Products & Services

Software for Sleeping Disorders. ResMed has introduced its new SleepTrack™ program which they say is the only software able to report whether a patient is being effectively treated by combining compliance data with efficacy and leak data. ResMed explains that the new program combines two components: technology and education. They say the patient first takes home the AutoSet®T, an auto-titration system, which records critical data such as mask leakage, dates and hours used, and the apnea/hypopnea index. Later, as determined by the doctor, they say the patient returns to the sleep lab where the data are transmitted directly from the AutoSetT to the doctor's computer using ResMed's proprietary AutoScan™ software. For more information from ResMed, circle number 195 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/



Digital EEG System. Bio-logic System Corp has introduced Ceegraph Netlink, the newest member of the Ceegraph product line for EEG, epilepsy monitoring and ambulatory recordings. According to Bio-logic, this new product incorporates the latest technological developments in networking, remote communications, and signal processing. Company press materials describe the Ceegraph Netlink as having unique capabilities, including an innovative amplifier system offering high signal quality and ultra-high signal resolution, an Ethernet network interfacing between the amplifier and the computer, allowing extended distances between the patient and the monitoring system without signal degradation, an option between a tower or a laptop computer without disparity in functionality, and remote controls on the headbox for bedside operation. Bio-logic says the system also offers flexible monitoring options with 40 channels, including options for 8 DC channels, built-in pulse rate and S_{aO_2} and a body position monitor. For more information from Bio-logic System Corp, circle number 196 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

POC Analyzer. Nova Biomedical has received Food and Drug Administration Clearance for point-of-care (POC) testing for their new Stat Profile pHOx® Plus Analyzer. The company says the new device integrates the key ten test critical care menu into a compact instrument and goes beyond the standard blood gas menu of pH, P_{CO_2}, and P_{O_2} to offer oxygen saturation ($S_{O_2}\%$), hematocrit, and hemoglobin. Nova Biomedical also says the pHOx Plus incorporates glucose, sodium, potassium, and a choice between ionized calcium or chloride to provide the essential test menu for the ICU, OR, ED, or stat laboratory. According to product press materials, the device also uses a liquid-only calibration system and offers an automated quality control system on a single onboard control cartridge. For more information from Nova Biomedical, circle number 197 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

Nasal CPAP Mask. Hans Rudolph Inc has designed a nasal CPAP/Bi-level mask for obstructive sleep apnea therapy. According to the company, the mask is soft, light, and low deadspace with multiple face sealing options to help ensure a good seal and a comfortable fit. Company press materials describe three optional sealing surfaces: a soft silicone rubber mask flange, the Ultimate Seal™ gel accessory, and the company's new Comfort Seal™ foam accessory. Hans Rudolph also says the device offers an adjustable nose strap which allows for additional custom fitting and forming to the patient's nose contour. For more information from Hans Rudolph Inc, circle number 198 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

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Calendar of Events

Date	AARC & Affiliates Programs	Contact
March 14–16	22nd Annual Tahoe Conference, **Lake Tahoe, CA**	CSRC Greater Bay Area Chapter, (925) 866-6643; www.cssrc.org
March 30–April 1	Tennessee Society for Respiratory Care's State Convention; **Franklin, TN**	Patti Joyner, (800) 473-6523, ext. 3101, paj2266@cs.com
April 4–5	USRC 2001 Conference; **Ogden, UT**	Utah Society for Respiratory Care, peberele@weber.edu
April 9–11	North Dakota Society for Respiratory Care's Annual Educational Symposium; **Fargo, ND**	Michelle Sele, (701) 234-6777, michellesele@meritcare.com, www.ndsu.nodak.edu/ndsrc
April 12–13	Rhode Island Society for Respiratory Care's 17th Annual Conference, **Newport, RI**	Wayne P. Foresti, (401) 769-8758, RIWPF732@aol.com
April 19–20	PSRC 4th Annual Eastern Regional Conference and Exhibition, **King of Prussia, PA**	Pennsylvania Society for Respiratory Care, Ann Cusano, (215) 646-7300, ext. 428, cusano.a@gmc.edu
April 25–27	Region II 28th Annual Meeting; **Covington, KY**	Jeff Jones, (800) 691-3041, #1, www.bright.net/~dsibb/reg2rc.htm
May 9–10	Maine Society for Respiratory Care's Spring Fling; **Lewiston, ME**	Roberta Crockett, (207) 262-2214
June 13–15	Illinois Society for Respiratory Care's 33rd Annual Convention; **OakBrook Terrace, IL**	Kelli DeBerry, (847) 981-3581, www.isrc.org
July 21–23	Management and Education Sections, Summer Forum; **Naples, FL**	AARC, (972) 243-2272, clay@aarc.org
Dec. 1–4	47th International Respiratory Congress; **San Antonio, TX**	AARC, (972) 243-2272, clay@aarc.org

Date	Other Meetings	Contact
March 16–17	UT Health Science Center and Wilford Hall USAF Medical Center's 6th Annual Respiratory Care Symposium; **San Antonio, TX**	UTHSCSA, Dept. of Respiratory Care, (210) 567-8850
May 12–14	Spring Sleep Seminar 2001, **Branson, MO**	Bill Rivers or Melinda Trimble, (501) 713-1272
Oct. 9-11	Cardiorespiratory Diagnostics 2001, **Las Vegas, NV**	Medical Graphics Corporation, Mari Orke, (800)950-5597, ext.444, www.medgraph.com

Call for Abstracts

2001 RESPIRATORY CARE OPEN FORUM

Early Deadline: May 31, 2001
Final Deadline: July 17, 2001

Notices

Notices of competitions, scholarships, fellowships, examination dates, new educational programs and the like will be listed here free of charge. Items for the Notices section must reach the Journal 60 days before the desired month of publication (January 1 for the March issue, February 1 for the April issue, etc). Include all pertinent information and mail notices to RESPIRATORY CARE Notices Dept, 11030 Ables Lane, Dallas TX 75229-4593

Scheduled Professor's Rounds 2001

Program #1 Taking the Mystery Out of Ventilator Weaning for Children—Peter Betit BS RRT FAARC; Host Richard D Branson BA RRT FAARC—Video March 13 Audio April 10

Program #2 Pulmonary Rehabilitation: Standard Care for Chronic Lung Disease Patients—Trina Limberg BS RRT; Host Thomas J Kallstrom RRT FAARC—Video March 27 Audio April 17

Program #3 Noninvasive Ventilation: The Latest Word—Dean R Hess PhD RRT FAARC; Host Richard D Branson BA RRT FAARC—Video April 24 Audio May 29

Program #4 Patient Education for the Asthmatic—Tracey Mitchell RRT; Host Thomas J Kallstrom RRT FAARC—Video May 22 Audio June 19

Program #5 ARDS: The Disease and Its Management—Leonard D Hudson MD; Host David J Pierson MD FAARC—Video June 26 Audio July 17

Program #6 New Respiratory Drugs: What, When, and How—Joseph L Rau PhD RRT FAARC; Host Patrick J Dunne MEd RRT FAARC—Video August 14 Audio September 11

Program #7 Invasive Ventilation: The Latest Word—Richard H Kallet MS RRT; Host Richard D Branson BA RRT FAARC—Video September 25 Audio October 16

Program #8 Test Your Lungs-Know Your Numbers-Prevent Emphysema—Thomas L Petty MD FAARC; Host David J Pierson MD FAARC—Video October 23 Audio November 20

Helpful Web Sites

American Association for Respiratory Care
http://www.aarc.org
— Current job listings
— American Respiratory Care Foundation fellowships, grants, & awards
— Clinical Practice Guidelines

National Board for Respiratory Care
http://www.nbrc.org

RESPIRATORY CARE online
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Asthma Management Model System
http://www.nhlbi.nih.gov

Keys to Professional Excellence
http://www.aarc.org/keys/

Committee on Accreditation for Respiratory Care
http://www.coarc.com

The National Board for Respiratory Care—Examination Fees for 2001

Examination	Examination Fees
CRT	$190 (new applicant) $150 (reapplicant)
Perinatal/Pediatric	$250 (new applicant) $220 (reapplicant)
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Authors in This Issue

Barnes, Thomas A .227
Bates, Peter W .279
Bishop, Michael J .234
Branson, Richard D .232
Chang, David W .282
Chatburn, Robert L .273
Chatmongkolchart, Sunisa .248
Currie, Donna .243
Dahm, Cornelia .278
Durbin Jr, Charles G .229
Finch, Derel .280
Giacoppe, George N .281
Hess, Dean R .248
Hussey, John D .234
Kacmarek, Robert M .248
Kester, Lucy .238
Lakshminarayan, S .234
Lands, Larry C .263
Lanni, Harold A .274
Liben, Stephen .263
Logemann, Jerilyn A .243
MacIntyre, Neil R .267
Massey, Lewis .234
Michalowski, Piotr .234
Moen, Suellen G .243
Mullahoo, Krishna .263
Noblett, Kenneth E .275
Orens, Douglas K .238
Peruzzi, William T .243
Seller, Lori .263
Serrano, Thomas .276
Smith, Stephen G .274
Sottiaux, Thierry M .255
Stoller, James K .238

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119	120	121	122	123	124	125	126	127	128	129	130	131	132	133	134	135	136
137	138	139	140	141	142	143	144	145	146	147	148	149	150	151	152	153	154
155	156	157	158	159	160	161	162	163	164	165	166	167	168	169	170	171	172
173	174	175	176	177	178	179	180	181	182	183	184	185	186	187	188	189	190
191	192	193	194	195	196	197	198	199	200								

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Authors in This Issue

Barnes, Thomas A	227
Bates, Peter W	279
Bishop, Michael J	234
Branson, Richard D	232
Chang, David W	282
Chatburn, Robert L	273
Chatmongkolchart, Sunisa	248
Currie, Donna	243
Dahm, Cornelia	278
Durbin Jr, Charles G .	
Finch, Derel	
Giacoppe, George N .	
Hess, Dean R	
Hussey, John D	
Kacmarek, Robert M .	
Kester, Lucy	
Lakshminarayan, S . .	
Lands, Larry C	263
Lanni, Harold A	274
Liben, Stephen	263
Logemann, Jerilyn A	243
MacIntyre, Neil R	267
Massey, Lewis	234
Michalowski, Piotr	234
Moen, Suellen G	243
Mullahoo, Krishna	263

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